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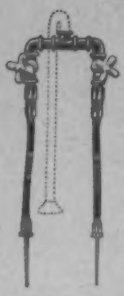

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THE
LARYNGOSCOPE.

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ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

GRANULOMA VENEREUM: GENERAL DISCUSSION WITH REPORT OF A CASE OF LARYNGEAL INVOLVEMENT.*

DR. ROBIN HARRIS, Jackson, Miss.

Definition: Granuloma venereum is a chronic, progressive granulomatous ulceration caused by the encapsulated bacillus of Donovan, affecting the skin and mucous membrane, usually beginning about the genital region and having no tendency to spontaneous healing. It is self-inoculable, affects both sexes, all races, occurs in most of the countries of the world, and yields to treatment with antimony and potassium tartrate.

Fernandez[1] defined it as "an infectious, contagious disease, self-inoculable, very frequently of venereal origin, which especially affects the skin and mucosa, and at times the general condition; in these latter cases a chronic ulcerous, vegetative and extensive evolution occurs, with a liquid secretion of a certain fetidness, in which one tends to acknowledge as an etiological factor the germ described by Donovan and Siebert, whether observing it to be free or involved in the cells which form granulomatous nodules which distinguish the process."

H. Aranjo de Sousa[2] thought that it affected only the external genital organs, and gave the following definition: "Venereal granu-

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loma is an ulcerous or vegetative dermatosis caused by the Calymmatobacterium granulomatosis, affecting generally the external organs of the genitalia and the perigenital zone."

Another good definition was given by Schochet[3]: "Granuloma inguinale is a mildly contagious infection of disputed etiology, characterized anatomically by a replacement fibrosis with secondary sclerosis and associated ulceration of the overlying tissues, a diffuse perivascular round-cell infiltration, and a formation of granulomata without caseation or giant-cell formation."

History: This disease was first brought to the attention of the medical world by McLeod[5] in his "Precis of Operations Performed in the Wards of the First Surgeon, Medical College Hospital, During the Year 1881", published in 1882, in which he described the disease as a serpiginous ulceration, but he did not give it any specific name.

Conyers and Daniels[6], in 1896, were the first to describe it as a distinct disease and called it a lupoid form of so-called groin ulceration, saying it was the most striking cutaneous disease affecting the pelvic region occurring in British Guinea at that time. Even at that early date they separated it distinctly from any disease of the lymph glands, and stated that it did not impair the general health. They were not convinced either that it occurred only in the negro race or that it was found exclusively in the pelvic region. They detailed nine cases, and the descriptions and types are exactly as those seen in our own country today.

Galloway[7], in 1897, called the disease ulcerating granuloma of the pudenda, and at that time reported a case in London.

Dempwolff[8], in 1898, found among the Papuans and Melanesians of New Guinea the same disease recognized by Conyers and Daniels[6], and gave it the name of granuloma contagioso.

Maitland[9], in 1899, applied the name chronic venereal sores.

From this time on I find no literature on the subject until 1905, when Donovan[10], in writing on ulcerating granuloma of the pudenda, was struck by the similarity of appearance of the lesions in the mouth of a ward boy to those occurring in fish suffering from microsporidia. Scrapings were taken and intracellular bodies described by him were designated parasites.

In 1907, J. M. H. MacLeod[11] reported the case of a soldier in England, who had been stationed in India, this being the first case in which X-ray was used as a method of treatment.

The next year, Sequiera[12], in writing on granuloma inguinale tropicum in a negro man with lesions at the left angle of the mouth and the right thigh, used X-ray treatment and obtained healing.

Row[13] found the presence of "eosin bodies" in the cell cytoplasm in cases of Oriental sore seen in Bombay in 1909. These eosin bodies may have been Donovan bodies and the disease not typical Oriental sore.

Jackson[14], in 1911, described an ulcerous disease of the genitals and perineum, which he had been seeing in the aboriginals in Australia since 1882. He at first thought that this disease was a form of syphilis occurring in the blacks, and for this reason it had been given the name of "black pox".

In 1912, Aragao and Vianna[15] reported a case of granuloma venereum, and in 1913 they stated that this disease resisted any and all forms of treatment, local and general. At this time they were using salvarsan, mercury and iodids, caustic preparations and X-ray. The authors had obtained no consistently good results from any of these treatments, and began the use of tartar emetic intravenously because of its universally good effects in cutaneous leishmaniasis (Oriental sore).

Grindon[16], in 1913, was the pioneer in this country. He had observed three cases in the previous eight years in St. Louis.

Symmers and Frost[17], in 1920, made a thorough study of two cases in Bellevue Hospital and were the first to describe the Donovan bodies in cases occurring in this country.

Name: Granuloma venereum has been applied to the disease in question by de Sousa[2], and Aragao and Vianna[15] in Brazil; Beatti[18], Fernandez[1], and Pasadas and Roffo[19] in Argentina; Silva[20] and Clement[21] in France; Cuthbert[22] and Bonne[23] in England. Ulcerating granuloma is the name that has been given by Donovan[10], and applied also by Cumming[24], Driver[25], and Sabella and Wise[26]. Other names include ulcerating granuloma, applied by de Matta[27] in Venezuela, Darwent[28] in Trinidad, and others in America and England. Granuloma pudendi tropicum by MacLeod[11]; tropical granuloma by Winfield[29]; granuloma inguinale tropicum by Sequiera[12] in England, Choyce[30] in England, and others in the United States. Granuloma inguinale is the name applied by most writers in this country, at least 20 giving it this designation; also Cole, Miskjian and Rauschkolb[31] in Germany, Pijper[32] in South Africa. Granuloma inguinale is possibly not the best name since lesions in this disease may be, and often are, found in various locations over the body, both on the skin and mucous membrane. Ulcerating granuloma of the pudenda is not applicable for the same reason. Ulcerating granuloma is hardly descriptive enough; and granuloma pudendi tropicum and granuloma inguinale tropicum indicate that it is a tropical disease, whereas at

the present time it is by no means confined to tropical climates. Lymphogranulomatosis inguinale is misleading, in that this is not a disease of the lymph glands in any sense. Granuloma venereum is used in this paper for the reason that in nearly all cases so far reported the disease began in the region of the genital organs, and nearly always followed promiscuous sexual indulgence.

Age: In the articles reviewed age was discussed in a few, and was specifically stated in most of the case reports. While the disease occurs most frequently in the adult, in the case of the Shattuck[31] child, circumcised at 1 month, the wound did not heal; Shattuck saw the child at the age of 2 years, and made a diagnosis of granuloma venereum. Heredity transmission has not been noted, but Gruzhit[33] mentioned a white woman observed by him, to whom was born a child covered with "pus pockets". The child died in the sixth month. The information was not sufficient to even surmise that this child was born with granuloma venereum, but de Matta[27] stated that rare cases had been observed in infancy. McLean[27] mentioned the disease in a male child of 6 years; and Sabella[27] a girl of 6 years with an intact hymen. Schochet[3], in a review of 66 cases, found two under the age of 22 years; 15 between the ages of 20 and 30 years; 13 between 30 and 40 years; four between 40 and 50 years; and three above 50 years. Winfield[29] reported a white female of 18 years, Sargent[30] a black male, also age 18 years. The oldest case found was reported by Scott[34], age 67 years, in whom a suprapubic operative wound did not heal after eight weeks. Tartar emetic was given intravenously and complete healing took place in 30 days. We find, therefore, that this disease is rare under 18 years of age, as well as in the aged. It is most common from young adult life to middle age.

Race: In McLeod's[5] description of serpiginous ulceration there were mentioned three Hindoos and one native Christian. The Hindoos are a religious sect living in India, who belong to the Aryan race. Conyers and Daniels[6] reported three Barbadian negroes, five negroes and one native of the colony. Fraser[35] divided his 19 cases into five groups: Two negroes, nine half-breeds, one Hottentot, three Kaffirs and three Cape Malayans. Wolff stated that circumcized subjects, such as Arabians, have not been observed with the disease. In North Africa, in the French districts, it is observed only among Europeans.

From 195 case reports reviewed at this time, there were 135 negroes, 35 white, 14 half-breeds, three Kaffirs, three Hindoos, two Australians, one Cuban, one Peruvian, one Mongolian.

Horwitz[37] believed that race played no essential causative part, although many writers thought the negro race had greater susceptibility. A. de Matta[27] believed there was no race preference, but that if negroes did form the highest percentage it was undoubtedly due to their lack of knowledge of personal hygiene. Cole, Miskjian and Rauschkolb[31], in 1928, saw 50 cases, mostly negroes, and they believed the prevalence was largely due to filth.

Ross and Kaupp[38] incorporated in one report 13 white people from Portland, Ore. The disease in four was traced to the same source. Three of these had the same occupation and worked at the same place, while the fourth was the janitor in the building in which the disease was contracted. In all of the literature reviewed, this is

Fig. 1. Showing elephantiasis of the scrotum. From Conyers and Daniels.

the only instance in which the origin of more than one case has been traced to the same source, and these four white men may have been exposed to the same white woman. The authors made no statement as to whether the person, or persons, called the source of the disease was examined, but if four white persons having been exposed in the same house to the same disease, and at least four white persons contracted that disease, there is strong evidence that race plays no part in susceptibility. They have now had 30 patients suffering from granuloma venereum, 28 of whom were white, one negro and one Filipino.

Sex: Grindon[16] concluded that: "The disease is not mainly one of women in this country, as Manson stated it was in the tropics."

Fernandez[1] believed the disease was not more common in any one sex, although he found that several authors had insisted that it was more frequent in women. Schochet[3] believed that, it being a venereal disease, males and females should be equally liable to infection; but Galloway[7] and many others reporting from the tropics found that it was much more frequent among females. In the United States case reports, where sex was specifically stated, there were 86 males and 45 females. In foreign articles reviewed there were 43 males and eight females. Those having treated the largest number of cases, namely, Thierfelder and Thillot[39], and Johns and Gage[40], did not state the number of each sex; therefore, any statistical report that can be compiled does not give the true status. We believe that this

Fig. 2. Showing mouth lesions before treatment. Courtesy of Dr. J. A. McIntosh.

is a venereal disease and, with Schochet[3], we believe since it is venereal disease there is no reason why men and women should not alike be subject to it.

Geographical Distribution: The geographical distribution of this disease is from one end of the earth to the other, as cases are being reported from every country and zone, from the equator to Scandinavia. It is apparently common in India, Australia, Brazil, Argentina, Peru, Venezuela and other South American countries; Costa Rica and other Central American countries; China, Italy, Spain, France, Germany, North Africa, the gold coast of Africa, South Africa and Norway. It has been noted in the United States from Boston to San Francisco; from Portland to Savannah; as far north

as Wisconsin, and as far south as southern Georgia and New Orleans. It is a notable fact that there are more patients suffering from this disease seen in the coast states and Great Lakes region. In the inland Rocky Mountain sections and the far western plains, no cases have been reported. While the disease has been seen in Kentucky, Tennessee, Arizona, Iowa and some other inland states, Kentucky, Tennessee and Iowa have river ports. Summing up the situation in the United States, it would seem that the disease travels from the sea ports towards the inland sections, and therefore must be a dis-

Fig. 3. Showing mouth lesions after treatment; also elephantiasis of the penis. Courtesy of Dr. J. A. McIntosh.

ease contracted from foreign countries. From this we gather that the people of no country are immune.

Incubation: Apparently little is known concerning the incubation period of granuloma venereum. Of the case reports and articles reviewed, only 11 authors mentioned a probable incubation period. Gage[41] reported one case where the period was one month, and Low and Newhan[42] a young man in which the primary papule appeared two days after he had had a casual connection with a black woman on the gold coast. Winfield[29] saw a primary lesion in a white woman six weeks after she had intercourse with a South American sailor, and one month after her marriage to a Russian, who had been in

the tropics but was perfectly healthy. Ross and Kaupp[28] found the incubation period in their climate (Portland) to be approximately the same as that of syphilis in the same climate, namely, three to five weeks. Two of their cases were given exactly as 24 days, and these were two of four cases contracted at the same source. McIntosh[43] found by experimental inoculation of a piece of granulation tissue from a spontaneous case, that on the forty-seventh day after inoculation, the wound for the first time was covered by a slough and oozed a small amount of characteristic exudate and smears from this contained typical intracellular Donovan bodies. This is the only definite and positive known period of incubation by experimentation.

Mode of Transmission: Just how granuloma venereum is transmitted from one individual to another is not known. Goodman[4] sug-

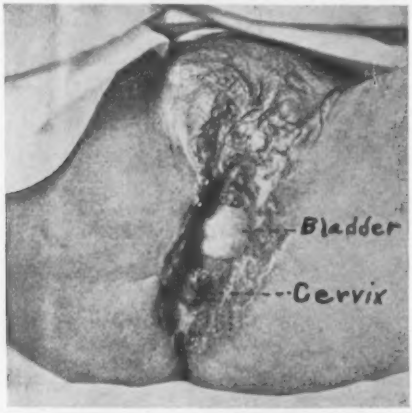

Fig. 4. Marked tissue destruction. From Rice.

gested that the contagion was probably direct and sexual, although an intermediate host, such as the pediculus pubis, was to be considered. He had two female patients with the disease, however, who had lived with their husbands eight months and two years, respectively, with neither husband contracting the disease. McRae[44] saw a white man, the father of six healthy children, who had extra-marital sexual intercourse once with a woman who had formerly lived in South America. The primary papule of what afterward proved to be granuloma venereum appeared eight days later. Theirfelder and Thillot[39] concluded that the infection occurred exclusively as a result of contact with infected individuals, chiefly by coitus. Most authors

apparently assume that transmission is by sexual intercourse. Low and Newhan[42] saw a man who had intercourse with a black woman only a few days before the primary sore appeared. Jackson[14] detailed the case of a white lad following cohabitation with an aboriginal woman. Many authors, namely, Aragao and Vianna[15] in Brazil, and Purcell[45] in Arizona; also Gruzhit[33] and Winfield[39] mentioned that the initial lesion appeared some time following illicit sexual intercourse. McIntosh[43] found that many exposures by coitus, both in the male and female, failed to transmit the disease to other individuals. These experiments were carried on in the Memphis General Hospital, under direct supervision and in negroes who were more or less dependable. He concluded that a break in the skin surface is necessary

Fig. 5. Rhinoscleroma seen in a foreign clinic. The resemblance was so striking that I had drawings made of these three cases for comparison with the after-effects of granuloma venereum in the larynx.

for the successful inoculation, or that the organism is infective only for susceptible individuals.

Cole, Miskjian and Rauschkolb[31] had the following to say concerning transmission: "We believe there is usually some relation to sexual contact, though in our series there were but one husband and wife treated concurrently. Many times we have had patients with lesions who gave us the history of regular marital relations, and yet the partner was free from the disease."

Types: Applehaus[46], Bergstrand[47], Goodman[4], Ross and Kaupp[38], and Randall, Small and Belk[48] have mentioned glandular involvement or enlargement. Most authors, notably Conyers and Daniels[6] among

the older writers, and Purcell[45] among the more modern, agree that glandular involvement plays no part in this disease. Careful perusal of the literature indicates that many authors in writing on malignant granuloma have in some instances given a good description of the subject at hand. Knowles[49] classified it as a new growth, while Conyers and Daniels[6] said it should not be called an ulceration, as, strictly speaking, it was a new growth. Nearly all writers are of the opinion that the disease is not a new growth, but a serpiginous ulceration. After a period of time, cicatricial contraction from efforts of Nature at repair may cause an elephantiasis-like enlargement. This type is prominently mentioned by McLeod[5]. The mass removed by him in one instance weighed 11½ ounces, another 2½ pounds.

Fig. 6. Rhinoscleroma seen in a foreign clinic. The resemblance was so striking that I had drawings made of these three cases for comparison with the after-effects of granuloma venereum in the larynx.

Among American writers, Fox[50] found 14 of his 15 cases to have had more or less elephantiatic enlargement. This enlargement involves the penis, scrotum and vulva, and is always associated with ulceration. It may be that there is an enlargement of the lymph glands early in the disease, as McIntosh[43] found that three days after transplant of tissue from a spontaneous case in the inguinal region, there was swelling of the inguinal glands, with very little pain or tenderness. Twenty-five days after the graft was planted there was slight swelling of the inguinal glands.

Gross Pathology: The lesion of granuloma venereum begins as a papule, the top of which later becomes a vesicle from which the

fluid escapes, the resulting ulceration spreading in all directions. It appears as an area, or areas, of partial denudation of epithelium, leaving a surface which is in some instances beefy-red in appearance, and in others covered with a thin, grayish membrane. If covered by a membrane, this may easily be scraped away, and beneath is a raw, bleeding surface. The surface of the lesion is granular in appearance, the latter being due to the lack of uniformity in the amount of granulation tissue present in different areas, producing a "piling up" of tissue in certain minute localities with depressions between. This irregularity of the surface had been compared to a relief map of a mountainous area. The size of the lesion varies according to the

Fig. 7. Rhinoscleroma seen in a foreign clinic. The resemblance was so striking that I had drawings made of these three cases for comparison with the after-effects of granuloma venereum in the larynx.

duration and rapidity of growth. The margin of the lesion is sharply defined and the tissue surrounding it is often elevated above the surface of the diseased area, giving an appearance of "piling up" or swelling of the surrounding healthy tissue. The depth of the lesion rarely exceeds a few millimeters, though in some instance not only the skin but also the subcutaneous tissues may be involved, leaving an area resembling a serpiginous ulcer.

When this is present there is no undermining of the edges. The ulceration does not ordinarily extend deeper than the deep fascia. There is no involvement of the lymph nodes except occasionally in the early stages, when a slight enlargement is present, and in these instances the enlargement of the glands disappears after a compara-

tively short time. It is possible that the regional adenopathy in the early state of the disease is due to an accompanying infection superimposed on the granulomatous area. The surface of the lesion is occasionally dry, but more often is productive of a thin, watery exudate with a characteristic, offensive, sour odor, and is frequently blood-tinged. This exudate in all probability contains the etiologic agent, as evidenced by the mode of spread of the lesion, which is always in the direction of the flow of fluid. The lesion spreads by either continuity or contiguity of tissue. That the lymphatic vessels in the infected area are involved is evidenced by the fact that a

Fig. 8. Skin lesions in the case of J. M.

moderate degree of elephantiasis occasionally occurs in the surrounding lesion. The most frequent location of the lesion is in the region of and surrounding the external genitalia. The pubis and groin are particularly favorite sites. In these regions the pubic hair is usually lost. The disease is not limited to the cutaneous regions, however, but often involves the mucous membrane of the glans penis, the vagina and the rectum. Neither is it confined to the areas proximal to the genitalia, but occurs on the abdomen, thighs, hands and various other portions of the body. In the mucous membrane of the mouth, pharynx and larynx, involvement is often marked and, while not common, has a characteristic granular appearance.

The extension of the lesion is quite slow, often requiring years to traverse a few centimeters. Complete spontaneous healing and epithelialization do not occur. Spreading takes place in the direction of the flow of the secretion, the latter being determined by gravity. Since few autopsies have been made, the effect of the disease on other organs of the body is practically unknown.

Histological Pathology: It is generally conceded that the microscopic picture of granuloma venereum is not sufficiently characteristic to render possible a diagnosis from study of a section alone. However, in the hands of a pathologist conversant with the histologic appearance of the lesion, the diagnosis might be strongly suspected. The outstanding features of the disease are the slowly-progressing

Fig. 9. Skin lesions in the case of J. M.

destruction and regeneration of the skin, and in some instances subcutaneous tissues, and the formation of granulation tissue. A section through the center of the lesion usually reveals islands of newly-formed epithelium, surmounting an area of young connective tissue. The latter consists of loosely arranged collagen fibres and connective tissue cells, in the meshes of which are numerous endothelial leukocytes, a moderate number of lymphocytes and a few polymorphonuclears. Many newly-formed blood vessels are present, and there is considerable swelling of the endothelial cells lining these vessels. The vessels themselves are dilated, and in many instances full of blood. No evidence of hemorrhage into these tissues, however, is present, although a few writers, notably Fox[50], and also Gage[41], have

observed ruptured vessels with irregularly-shaped pools of blood and lymph. The existence of giant cells has been denied by the majority of investigators, though Bergstrand[47] reported the existence in a section from a lesion on the pharynx of giant cells which resembled Langhan's giant cells. He stated, however, that they differ to some extent from those usually found in tuberculosis and syphilis, in that the nuclei are larger and form rings which close around the whole cell. There is no evidence in the lesion of caseation or suppuration. The epithelium which occurs within the lesion in islands, separated from each other by areas of uncovered granulation tissues, consists of morphologically imperfect cutaneous tissue. The horny layer is absent, or it is not well formed, and the cells of the stratum mucosa

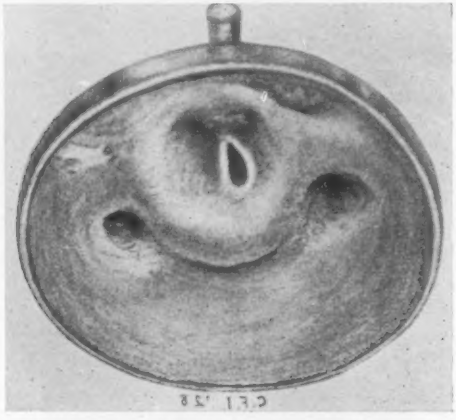

Fig. 10. A drawing of the larynx of J. M. just before the swelling had entirely subsided.

are poorly defined and swollen. There is an absence of the normal skin pigment, though Gage[41] observed a brown pigment occurring in clumps, just beneath the papillae of the epidermis, and in some areas more deeply situated. He suggested that this pigment bears a relationship to some type of cell, either connective tissue or endothelial cells present in this locality, and stated that it is probable that the pigment has been taken up by these cells from that which has been disassociated from the basal cells of the epidermis, or was hemosiderin resulting from repeated small hemorrhages that had taken place in these tissues. Only a few hair follicles and sweat glands remain, and in those remaining a thickening or swelling of

the cells is noted. Surrounding these follicles and glands a marked infiltration of round cells may be seen. Residua of follicles and glands almost completely replaced by sclerosing connective tissue is noted. At the margin of the lesion are finger-like projections of partially destroyed and regenerated squamous epithelium projecting from the normal skin surrounding the lesion. The more normal cutaneous tissue at the periphery has undergone hyperplasia of cells, and this hyperplastic tissue extends down under the edge of the granulation tissue. Beneath a normally appearing epithelium extending some distance out from the margin of the lesion there is present marked infiltration of round cells, indicating that the lesion extends further out than would appear on superficial inspection.

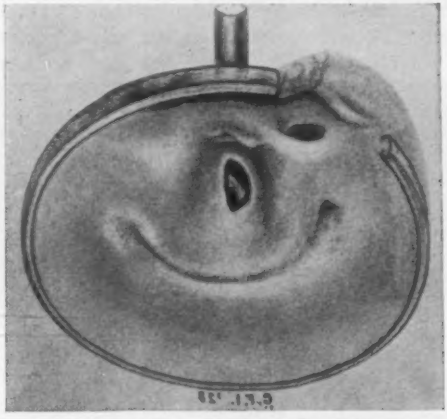

Fig. 11. The larynx of J. M. after all swelling was gone.

A very good account of the work done by investigators in the determination of the causative agent in granuloma venereum is given by Clement[21]. That the disease is of an infectious nature, there can be no reasonable doubt. The fact that it occurs in a majority of instances in the region of the external genitalia, and the fact that in numerous instances its inception may be traced to sexual intercourse, indicates not only the infectious but also the venereal nature of the disease. It is readily conceded that numerous instances of sexual contact with victims of the disease have failed to produce lesions in the healthy individual. This does not preclude the infectious nature, however, since in all probability an abrasion of the skin of the healthy individual is also necessary for inoculation. The over-

whelming preponderance of cases in which contagion or the disease has been traced to contact with foreigners, in whose country the disease is common, minimizes the significance of the comparatively rare instances in which exposure fails to result in infection. The response of the infection to its specific treatment is slight evidence also of the infectious nature. It is quite natural that a chronic lesion of the nature of granuloma venereum should have been ascribed from time to time by observers to tuberculosis, luetic infection, neoplasm or localized trophic disturbances. That the lesion is not tuberculous has been definitely proved by the uniform inability to demonstrate tubercle bacilli in the involved tissue or its exudate, by the inability of the experimenters to produce tuberculosis in guinea pigs by inoculation, and by the fact that the histologic picture of the lesion differs materially from that of tuberculosis. Although one or two investigators, Goodman[4] and Weinberg[51], have reported the find-

CHART ON DURATION OF GRANULOMA VENEREUM

	80 CASES	AVERAGE DURATION	0-1 yr.	1-2	2-3	3-4	4-5	5-10	10-20	20 up
U.S.A.	67	3.6 yrs.	23	15	10	10	6	8	4	0
Foreign Countries	25	6.4 yrs.	7	5	1	1	2	6	3	0
Foreign in U.S.A.	8	10.1 yrs.	0	1	1	0	0	1	1	1

ing of spirochetes in the lesion, the syphilitic nature of the disease can be definitely eliminated by the negative Wassermann reaction in the majority of instances, and by the absolute failure of the lesion to respond to antisyphilitic treatment. Although McIntosh[43] and others have observed mitotic figures in the lesion, the lack of uniformity and the apparent paucity preclude the probability of the lesion being neoplastic. Again, its response to specific treatment contradicts its neoplastic nature. The transmission of the disease by transplanting a small section of tissue from a spontaneous lesion into a healthy individual, as accomplished by McIntosh[43], rules out the possibility of a localized trophic disturbance as an etiologic factor. The only organism so far discovered which can be seriously considered as a specific infectious agent is that first mentioned by Donovan[10], in 1905, and later studied by Flu[52], Castellani[53], Aragao and Vianna[15], McIntosh[43] and others. This organism has almost universally been called "Donovan body", after its discoverer, although Aragao and Vianna[15] suggested the name calymmatobacterium granulomatis.

Donovan[10] came upon the discovery by the striking resemblance of a lesion of granuloma in the mouth of a patient to that in fish suffering from microsporidia. From scrapings from the deeper parts of this lesion the oval bodies were found within the epithelial cells of the stratum malpighii. Donovan bodies are pleomorphis, coccoid to bacillary in shape, nonmotile, nonsporulating, from 0.5 to 2 microns in diameter, Gram negative, and staining readily with Wright's stain. In their natural habitat they are encapsulated and found within epithelial cells. They grow best on Sabouraud's medium, on which they appear as small, translucent, rounded, dome-like colonies with smooth margins, from 0.5 to 2 m.m. in diameter. They will grow at room temperature, though body temperature is more conducive to growth. They grow both aerobically and anaerobically. Subcultures grow well on blood serum, blood agar, nutrient agar and lemco agar. Gas is produced in anaerobic growths. Methemoglobin develops around colonies on blood agar plates. Acid is formed in litmus milk, dextrose and mannite. Milk is coagulated. Gelatin is not liquefied. Donovan bodies have been recovered fairly uniformly from cases of granuloma venereum by practically all recent investigators. McIntosh[43] has found them in 14 of 15 cases; Fox[50] in 11 of 15 cases. McIntosh[43] has probably done more than any other investigator to substantiate the belief that the Donovan body is the specific causative agent in granuloma venereum. He has successfully transmitted the disease to a healthy individual by implantation of a section of granulation tissue. Furthermore, by culture of a portion of tissue from this same lesion he recovered Donovan bodies, and no other organism. Although this is strong evidence, it cannot be definitely stated that some virus was not present in the tissue grafted. Donovan bodies were obtained in pure cultures from the lesions occurring at the site of the graft. A typical lesion of granuloma venereum has not been produced by injections of pure culture of Donovan bodies, and until this is done, and the Donovan bodies again recovered from the lesion so produced, the postulates of Koch have not been satisfactorily met.

Fernandez[1], in summing up, had the following to say: "This study has also been made difficult by having been unsuccessful in inoculating human material to transmit the disease to different animals, such as guinea pigs, monkeys, rats, rabbits (experiments of Conyers and Daniels, 1896; LeDantec, 1905; Cleland, 1909; Steel, 1912; E. Martini, 1913; Aragao and Vianna, 1913; de Sousa, 1914; and Lynch, 1921). The only results obtained were either fatal septicemias, gangrenous forms or local nodules which promptly were reabsorbed.

Neither have they been able to reproduce the disease by inoculating men with calymmatobacterium."

Duration: This disease has usually existed for a period of from a few months to several years before a diagnosis is made. In some localities, where the disease is very common, some doctors have become adept at making the diagnosis. In the United States, the average duration is 2.6 years in 87 case reports in which the duration is mentioned. Of this 87, 33 had existed less than a year, 15 from one to two years, 10 from two to three years, 10 from three to four years, six from four to five years, eight from five to 10 years, four from 10 to 20 years, and none above 20 years.

In the United States, "the soldier of fortune" is taken separately; by this term is meant the sailors and travelers, not the highest type, who are in and out of foreign countries. Five of these had an average duration of 10.1 years, but this disease in one had existed for 25 years, and in another, 15 years.

In 25 foreign cases the average duration was 4.4 years. Duration less than one year was seven; one to two years, five; two to three years, one; three to four years, one; four to five years, two; five to 10 years, six; 10 to 20 years, three.

Distribution in the Body: Granuloma venereum begins as a papule, usually on the glans penis or foreskin in the male; in the vagina or in the labia in the female. This papule ulcerates and extends in all directions, usually early involving the pubis, groin, and later the perineum, scrotum and rectum. The extension appears to be particularly in the direction where moisture and warmth are greatest. Aragao and Vianna[15] described the lesions, seven in all, of various sizes, spread over the penis, scrotum and abdominal regions. In Rice's[54] case there was complete necrosis of all the vulval structures, the urethra, vagina, perineum, levator muscles, triangular ligaments, rectum with sphincter muscles, up to Douglas' pouch, and the bladder was exposed. Donovan began by saying: "A ward boy was admitted for this complaint in his mouth." He makes no further statement, and one is led to believe that finding lesions of this disease about the mouth was nothing out of the ordinary in India. Extra-genital involvement is by most recent authors considered to be a secondary manifestation. Sequeria detailed involvement of the angle of the mouth and inguinal region. Willmott[35] stated that Ross, of Virginia, reported a case of involvement of the nose and larynx. Bergstrand[47] found lesions beginning in the pharynx which extended to the larynx, soft palate and pillars of the fauces, which later involvement of the inguinal glands. He found acid-fast bodies in crumbled cells

and granula, which in his description resembled to some extent other descriptions of the Donovan bodies. The groin gland involvement may have been a different affection, but is connected by the author with the disease that began in the pharynx. He described these bodies as being within monocytes and also lying free. If this be a true case of granuloma venereum, it is the only one on record that began with throat involvement. In a personal communication to Cole, Miskjian and Rauschkolb[31], Dr. J. B. Shelmire, of Dallas, Tex., wrote of a man in the seventh year of his disease, with ulceration involving the penis, scrotum, thighs, pubes and lower lip. John and Gage[40] recovered Donovan bodies in one instance from a lesion on the sole of the foot; another case presented lesions involving the chest, cheek, lips, gums and roof of the mouth. Sidlik[56] found Donovan bodies in scrapings from lesions on the mucous membrane of the mouth. Taussig[57] found Donovan bodies in secretion from a secondary lesion on the lower lip. In one of six cases, Theirfelder and Thillot[39] recovered microbacterium granulomae in pure culture from an abscessed cavity; healing followed antimony and potassium tartrate injections, but three months later the patient returned with new lesions on the face and right submaxillary gland region. A second case presented granular ulcerations on the left labium minus, on the forehead, and beneath the angle of the right lower jaw; while a third case developed lesions about the right ear and a very large area over the mastoid. This latter extended inside the ear canal to beneath the lower jaw and the right side of the neck. In a fourth case there were ulcers on the right side of the thorax and over the right clavicle. The fifth case, on the back over the right kidney region; and in the sixth and last case, on the right index finger. In all of these the ulceration developed in association with long, persisting, small, primary genital lesions. Hunter[58] reported lesions of the groin, perineum and lip in a case described by him. This patient had been treated for syphilis with no improvement. Donovan bodies were recovered from all lesions, including the one on the lip. The patient was extremely hoarse and could not open the mouth; therefore, visual examination was not made of the mouth or throat until tartar emetic had been administered. Following this treatment, however, dysphagia, the ability to take food, and the voice improved rapidly and markedly. Then there was found cicatricial stenosis of the palate, the palate being adherent to the posterior pharyngeal wall, and the epiglottis had either disappeared or was included in the cicatrix, leaving an oval opening into the larynx. There were no vocal cords. He described a view of the larynx as simply looking

down into a circular opening. The uvula and tonsils had disappeared.

Case Report of Laryngeal Involvement: J. M., negro male, age 28 years, and divorced, was admitted to the U. S. Veterans' Hospital, No. 88, May 6, 1926. He was an ex-soldier of the World War, having served in France, and was a native of Edgefield, Gransville and Trenton, S. C. He was strong, healthy, muscular, and physical examination was entirely negative except for the granulomatous lesions described below. Two years or more previous to admittance, his trouble began with a pimple on the prepuce. A few days or weeks later, he was not quite sure which, a red-bug bit him on the left groin, and from the resulting papule a small amount of discharge was constantly present for six months. This gradually grew larger, and while he was treated several times by various doctors, the ulceration gradually grew larger until at the time of admission there were several large ulcerations on the scrotum, perineum and anus, extending as high up as the coccyx behind, and at all points where the nates came in contact. The whole of the left inguinal region was a large ulcer, while the entire penis was free of ulceration except at the base on the left side. There were some small healed scars, indicating that the penis had not always been free from ulceration. Below the angle of the left jaw, on the side of the neck, there was an oval granular ulceration about 3 x 1½ inches in size. The day after admittance, the patient was sent to the ear, nose and throat department for examination, and came under my observation. He gave a history of having had a sore throat seven or eight months previously, but he was very positive that there was nothing wrong with his throat, at that time, and he did not want a throat examination. However, he was quite hoarse and on examination there was found an ulceration of the left tonsil. There were small, raised granulations and scar tissue extending from the anterior pillar to the left angle of the mouth, becoming less marked as the distance from the anterior pillar to the mouth angle increased. These small granulations were interspersed with slight fibrosis, which resembled syphilis, and a clinical diagnosis of syphilitic ulceration was made. The larynx was red and swollen, but no ulcerations were present. At this time the epiglottis was free of inflammation. The vocal cords were thickened and red, as were all the visible portions of the larynx. A note was sent to the ward surgeon with a tentative diagnosis of clinical syphilis, and a request for the return of the patient in 10 days for further observation. However, he was not seen again until July 16, 1926, at which time the ulceration in the region of the left tonsil had

improved, and the following note was made: "There is much swelling of the glottis, and this patient will bear watching for dyspnea. If dyspnea becomes apparent a tracheotomy is necessary." The patient was not seen again in the ear, nose and throat department until Oct. 27, 1926. At this examination there was found a scar in the region of the left tonsil. No tonsil was present. There were small spots of scar tissue from the left anterior pillar nearly to the left angle of the mouth. The laryngeal condition had improved and it was found that since July the ward diagnosis had been changed from syphilis to granuloma venereum, and the patient had been on treatment with tartar emetic. The ulceration on the neck and groins had improved markedly, but were not healed. He was discharged Oct. 27, 1926, as not in need of further hospitalization. On Dec. 21, 1926, he was readmitted to the same hospital and it was found that the ulcerations about the genital regions had recurred, as well as the neck lesion, and all of these lesions were just as bad as when the tartar emetic was first begun. The larynx was also found to be in all visible parts greatly swollen and much edema was present. The epiglottis was adherent to the base of the tongue and was ulcerated; the ulceration extending to the base of the anterior tonsillar pillar on the left side. There was a great deal of scar tissue in the region of the glottis, the vocal cords were not present and there was a small, slightly oval opening into the larynx. The whole upper part of the larynx was so edematous and swollen that all landmarks were obliterated, and the examiner simply looked down into a hole that was built up, so to speak, all around the outside of the hole, as an old well curbing or a crayfish nest. The patient was put on antisyphilitic treatment at this time, but each dose of salvarsan increased the dyspnea for several hours following administration. The condition of the patient did not improve in any manner under antisyphilitic treatment, and the treatment was again changed to tartar emetic intravenously (one dose), and here it should be noted that the tartar emetic had never been given at regular intervals. On March 16, 1927, the larynx was found to be perhaps a little more edematous than at any previous time, and the following note was made: "In view of the fact that the patient had great relief from his dyspnea about 10 days ago following a dose of tartar emetic intravenously, I would recommend that this patient stay in the hospital and receive regular treatment with tartar emetic intravenously every other day." On the following day, a specimen of tissue was taken from the region of the left arytenoid and the base of the epiglottis. This tissue was sent to Dr. J. A. McIntosh[43] for examination, who found moderate hyper-

plasia of the mucosa in some areas and ulceration in others; polymorphonuclear and round-cell infiltration of the submucosa. There was marked endothelial proliferation of the blood vessel lining and many new blood vessels, together with an occasional vein stuffed with polymorphonuclear cells. There were a moderate number of swollen, pale mononuclears with indefinite cytoplasmic inclusions which were considered to be Donovan bodies, and a diagnosis of granuloma venereum of the larynx was rendered. (At this time this was found to be case "J. M." of the McIntosh[43] report.) On March 18, 1927, the day following removal of the specimen from the larynx, dyspnea became much worse, and a trachetotomy was done, giving immediate and complete relief. There was apparently no extension of the granulomatous pathology in the trachea at the point entered. The patient then received tartar emetic intravenously, in doses ranging from 2 c.c. to 10 c.c. in 1 per cent solution on alternate days for six weeks, improving gradually. After a lapse of 30 days, treatment was begun again and continued for one month, at the end of which time, all skin lesions were completely healed. There was at this time no epiglottis present and the larynx was a mass of cicatricial tissue. The picture was somewhat as shown by the accompanying drawing, which was made about this time. The patient was given three doses of tartar emetic intravenously each month until discharged for transfer to the National Soldiers' Home at Dayton, O. At the time of discharge, all lesions had been completely healed for several months, the patient was thoroughly satisfied with his condition, and the breathing was so good through the tracheotomy tube that he expressed a desire to be allowed to go without further operation. No operation was done for the laryngeal stenosis.

Diagnosis: The diagnosis is made on a fairly characteristic clinical picture, and on the bacteriological findings of a specific organism. This is done by making smears from the exudate on the surface of the raw, ulcerated area. In this exudate will be found abundant large mononuclear cells, the protoplasm of which, on staining with Wright's or Giemsa's stain, will be found studded with the characteristic encapsulated bacilli originally described by Donovan. The lesion appears clinically as a light red, shiny mass of granulating tissue that bleeds easily and exudes a thin, light, serous or serosanguineous fluid and has a characteristic fetid odor. The healing, which takes place following the use of antimony and potassium tartrate intravenously, is indicative of the accuracy of the diagnosis, since after administration of from two to 10 doses marked improvement is usually evident. The diagnosis is often made by finding that the disease resists the

treatment for various other diseases, and then obtaining the careful history of the ulceration spreading from a small, primary, slightly itchy papule, on or about the genital organs. For example, Fernandez[1] takes into account the beginning and evolution in the genital organs, the seropurulent secretion with its characteristic foul odor, the aspect of the flat, slightly grainy or very granular ulcer; the ulcer not forming a continuous clean line, the absence of adenitis, and the findings of intracellular and extracellular Donovan corpuscles in the exudate.

Differential Diagnosis: Granuolma venereum and Oriental sore, or cutaneous Leishmaniasis, are alike in that there are ulcerations on the skin; and in each the larynx, pharynx and buccal mucous membrane may be involved. Antimony and potassium tartrate are specific for both diseases. They are unlike in many respects: Oriental sore occurs most frequently on the uncovered parts of the body, whereas granuloma venereum occurs most frequently about the genital region, and in warm, moist places. The temperature in the one is above normal, and in the other, normal. The mortality in Oriental sore, untreated, is high; in granuloma venereum, practically *nil.* Finding of Leishman bodies from scrapings of the ulcer is diagnostic of Oriental sore, while the Donovan bodies are characteristic of granuloma venereum. Tartar emetic usually cures Oriental sore in a few days, while it usually takes a period of months to complete a cure in granuloma venereum. Fernandez[1] classed Leishmaniasis as not a process of venereal origin, whereas the evidence is that granuloma venereum is a venereal disease. Oriental sore is more frequent in children, whereas granuloma venereum is more frequent in adults.

Granuloma venereum is probably mistaken for syphilis more often than any other disease and in many instances occurs with syphilis in the same individual. The majority of the cases seen by Lynch[59] were associated with syphilis. The characteristic appearance of the ulcer in granuloma venereum is different in most instances from the syphilitic ulcer. If the two diseases occur in the same person, the patient is usually treated for syphilis because of the positive blood Wassermann. In these cases the ulceration does not heal and one should always then be suspicious of granuloma venereum, and make smears properly stained for Donovan bodies. If found, a few doses of antimony and potassium tartrate will usually show improvement if infection with the Donovan bacterium be present. Syphilis is characterized by the enlargement of the lymphatic glands in contrast to no glandular involvement in granuloma venereum.

Clinically, granuloma venereum has often been confused with tuberculosis cutis. The finding of giant cells in the latter, together

with positive findings in animal inoculations, and the presence of the tubercle bacilli against the finding of Donovan bodies in the former is sufficient to differentiate the two diseases in doubtful cases.

In chancroid, the inflammation is markedly acute and painful, the ulcers are undermined, and it is usually complicated by inguinal adenopathy and responds rapidly to local treatment. Ducrey's bacillus is the causative agent in chancroid. In granuloma venereum, the process is chronic, painless, the ulcers are rather rolled out, there is usually no inguinal glandular involvement, never bubo formation, and the disease does not respond to local treatment.

Many articles have been written on malignant granuloma of the genital region. The clinical description in many of these case reports fits granuloma venereum better than it does carcinoma. Many cases of granuloma venereum have been diagnosed as carcinoma even by pathological section. In Sargent's [60] case, the diagnosis for months was cancer of the penis, with metastasis of the groin. Sections from both the penile and groin regions were pronounced carcinoma on two seperate occasions by a very competent pathologist. Sargent stated that a third biopsy, made after treatment with antimony and potassium tartrate had been instituted, and the sections cut perpendicularly to the skin surface, disclosed the true nature of the lesion. That the diagnosis may be confusing is well illustrated in a case reported by Best. The diagnosis was first syphilis; after a time it was changed to cancer. She was given narcotics *ad libitum* and told that she was going to die. After many months, the diagnosis was again changed to syphilis, and finally the correct diagnosis was made and antimony and potassium tartrate given, resulting in the lesion completely healing in four weeks.

In this climate yaws may be ruled out, since apparently it is entirely a tropical disease and, too, yaws yields readily to antisyphilitic treatment.

In blastomycosis the lesions have a tendency to crusting and the exudate is thick and purulent. Microscopically, budding, yeast-like cells may be found in the discharge, and in cultures there is the formation of a mycelium resembling that of an oidium.

Lymphogranulomatosis, or inguinal lymphogranulomatosis, is not to be confused with granuloma venereum, although Destefano and Vaccarezza have reported cures in a large series of cases by antimony and potassium tartrate.

Because elephantiasis in a moderate degree is often present in the disease under consideration, filarial infections must be excluded. This may be done by examining the blood in fresh night specimens.

Conyers and Daniels[6], in Case 1, found filariae complicating granuloma venereum. However, filarial infection in the United States is rare.

In the mucuose membrane, rhinoscleroma resembles granuloma venereum more than any other disease except, perhaps, syphilis. While rhinoscleroma is not common in this country, neither is granuloma venereum, and the two diseases must be differentiated. They both run an extremely chronic course, but in rhinoscleroma the effect is more a thickened mucous membrane with a crusty, tenacious, fetid exudate. The lesion is best described as being indurated and very hard. While rhinoscleroma is frequently primary in the larynx, mouth or nose, granuloma venereum in these areas is usually secondary to the genital lesion. In granuloma, the swelling in the mucous membrane is marked. There are some ulcerations and a great deal of edema. The bacillus of von Frisch is found in cultures from the discharge in rhinoscleroma, and closely resembles in many respects the bacterium of Donovan. While Figi and Thompson[62] reported good results in the treatment of rhinoscleroma by radium, most authors conclude that no treatment either improves or arrests it. Granuloma venereum yields to treatment with antimony and potassium tartrate.

Symptoms: The symptoms of granuloma venereum are largely negative, in that there is complete absence of fever, chills, leukocytosis, throbbing, lassitude, aching and other concommitants of acute infectious processes. Wassermann tests are negative and the ulcer does not heal after administration of arsephenamin, as does chancre. The objective symptoms include the presence of an ulcer which began with a small, slightly itchy papule.

Except for the characteristic fetid discharge, the patient does not seem to be affected by the morbid process and there is no pain. When two raw surfaces in the genital region rub together there is a peculiar gait, no different from that of other diseases in this area. The symptoms in location other than the genital area are very much the same as would occur in cancerous or syphilitic ulceration.

Prognosis: Untreated granuloma venereum may go on for years with no constitutional symptoms other than the secondary anemia, and the consequent weakness. The prognosis is good as to life, and most individuals with the disease "carry on" with very little inconvenience or discomfort. Death occurred in a female reported by Rice[54] in which there was extensive ulceration, with exposure of the viscera. Properly treated, recovery takes place in practically all instances.

Prophylaxis: Very little can be said of prophylaxis other than to agree with Schochet[3]: "The disease should be placed on the list of reportable infections and patients should be placed in hospital quarantine. Segregation should be compulsory, as most of the unfortunate individuals belong to the lower strata of society, and even with extensive lesions sexular relations are indulged in. As these cases are rebellious to treatment, and painful, it is not uncommon that many of these patients desert." Placing this disease on the list of reportable infections would probably do more than any other one thing to stimulate members of the medical profession throughout the world to be on their guard in making a diagnosis.

Treatment: In the early history of this disease, surgery—radical surgery—was found to be the best treatment. Antiseptics were of little use even in the days of McLeod[5]. Strong caustics in conjunction were used by him, but he depended on removal of large amounts of tissue as a cure. He mentioned making flaps from the thigh to cover at least a part of the area from which all skin was removed and thoroughly curetted. Conyers and Daniels[6] regarded ordinary antiseptic dressings as having no influence on the growth, and antisyphilitic treatment was of no avail. They depended for success on complete removal of the growth, followed by scraping with the sharp spoon, and the use of such caustics as chlorid of zinc, nitric acid and carbolic acid, extensively employed in the recurrent spots. Freidlander[03] as late as 1913 found the only effective treatment to be surgery. J. M. H. MacLeod[11] appears to be the first to conceive the idea of employing Roentgen rays in the treatment of granuloma venereum. The patient who served for this application of X-ray was a soldier in the Royal Army Medical Corps, who had acquired the disease in India in 1903. The treatment was begun in October, 1906, and lasted for several months. Fifteen small doses at weekly intervals effected a cure. Based on MacLeod's[11] experience, Sequiera[12], in 1908, adopted this method of treatment over a period of eight weeks, seven applications being given for multiple ulceration in the inguinal region, dorsum of the penis, and left angle of the mouth. Recovery was complete. In the same year, 1908, 37 patients were similarly treated in the General Hospital of Madras with excellent results. This information was given by de Sousa[2] from the hospital records, and at the same time he reported his own case treated by the application of "5-H for 10 minutes at intervals of two weeks." The patient complained of pain and pruritis for some days after the first application. After four treatments, the ulceration was completely cicatrized. This method of treatment was also employed by

McLean[64], of the General Hospital of Brisbane, Queensland, Australia, in 1911. Carty[65] effected a cure by X-ray when there was extensive exuberant granuloma of both inguinal regions complicated by extensive bone changes in the pelvis, for which latter orthopedic treatment had been given for a period of one year. Whether the bone changes were thought to be in any way connected with the granuloma was not stated, but both troubles were cured by the application of X-rays. Fox[50] found in one case that the disease disappeared almost completely after six exposures of unfiltered irradiation had been given (a total of 4¼ skin units). Many other writers found that Roentgen rays improve the disease in some instance, and especially has this been true in South America. Many types of treatment have been used but mention here will be made largely of those that have been of some benefit. Aranjo de Sousa[2] advocated combined X-ray and intravenous administration of antimony and potassium tartrate as the surest method of cure. Antisyphilitic treatment had been universally found to be of no avail. Mercurochrome intravenously was used successfully by Willmott[55] (Case 4). Twenty doses (15-20 c.c.) were given at weekly intervals. The lesions were healed completely when examined three months after treatment was ended. The same patient had had tartar emetic intravenously for a period of several weeks just before the mercurochrome treatment was begun. McIntosh[43] used gentian violet intravenously with some improvement. Choyce[30] mentioned that "Mr. Pinch said that the last case of the kind which he saw in India was in the person of an Englishman, an occurrence which was very uncommon. It took a long time for the patient to get well, though the case was seen early. It was ultimately cured by scraping, followed by zinc ionization." Vaccine treatment has been used by Sidlik[56], Aragao and Vianna[15], McIntosh[43] and others, with no apparent good effect; however, Goldzieher and Peck[66] (Case 5) used tartar emetic for six months, followed by vaccine treatment, "which did more toward healing than did tartar emetic."

New preparations of antimony have been used successfully by several since 1922. Randall[67] used sodium and antimony thioglycholate and triamid of antimony thioglycholate prepared by Prof. Abel, of Johns Hopkins University, with good results and complete absence of the disagreeable symptoms following the administration of antimony and potassium tartrate. Randall, Small and Belk[48] used antimony thioglycholate in Case 12 of their series. Shattuck and Little[68] used both of the above-named drugs, both intravenously and intramuscularly, without bad effects, and their results were good.

Ross and Kaupp[38] also used these drugs, but in a recent personal communication they stated that substitutes for tartar emetic are not sufficient. These new preparations, if found to be just as efficacious as antimony and potassium tartrate and without its toxic effects, should be given a thorough trial.

Aragao and Vianna[15] began the intravenous administration of antimony and potassium tartrate, because of the good effects of this drug in cutaneous Leishmaniasis. These injections gave very satisfactory results as they made possible rapid cure of the patient. The drug was administered in a solution of 1 per cent, sterilized by filtration. The dose for each injection, given on alternate days, was between 60 and 120 c.c. of 1 per cent solution; a series of 10 injections of 100 c.c. each being at times sufficient to obtain complete recovery. There was generally very little inconvenience in connection with this treatment. If cough occurred, the injections were interrupted. After the injections the patient occasionally suffered chills, nausea and, more rarely, joint pains. All of these, however, rapidly disappeared, allowing the treatment to be continued. The patients sometimes complained of pruritis and formication in the ulcer. The granulations in the affected tissues very rapidly disappear; with the disappearance of the granulations, cicatrization took place rapidly, beginning at the borders of the wound in frank epithelial proliferation; in the central part as well there were small islands of this tissue from which expansion took place centrifugally.

Bonne[23] gave very large doses of tartar emetic, starting with 80 c.c. and increasing to 150 c.c. (percentage not stated). After the regular treatment, he gave four injections at irregular intervals, the last of which contained three grains of the drug. In spite of the large doses given by some, Johns and Gage[40] had two deaths following doses of 18 and 20 c.c. of 1 per cent solution per injection. The authors stated that both fatalities presented all of the indications given in the textbooks for tartar emetic poisoning, namely, multiple punctate hemorrhages, no postmortem clotting of the blood, and acute yellow atrophy of the liver. They also suggested that in view of the fact that tartar emetic is said to be a culminative drug, that proportionately few doses cause almost complete disappearance of the etiologic agent, and in view of the probable damage to the liver by long-continued sublethal doses, that interrupted courses of treatment be given instead of the usual almost-continual plan. Tartar emetic has been applied locally and, in some instances, an improvement resulted. Also, it has been given intramuscularly. The tissue reaction is so great that this method was early abandoned. It has

been given by mouth, but the most approved method is by intravenous injection of much smaller doses than were used by the authors originating this treatment. In the use, however, of tartar emetic intravenously, great care should be taken to have the needle in the veins before the injection is made, as infiltration of the tissues outside the vein is exceedingly painful, causes a great deal of reaction, and may even slough much the same as arsphenamin under the same conditio: s.

Theirfelder and Thillot[39], while not observing at any time severe symptoms of poisoning, believed that a course of treatment should not exceed 20 to 23 injections without interruption, as otherwise paralysis and cachexia might occur.

In 1919, Lynch[59] stated that no successful treatment had been devised, and all of his cases had died except one, and in this the infected member had been amputated. In 1921, the same author found that surgery and tartar emetic were effective, and that all his recent cases had been cured with tartar emetic.

Where granuloma venereum exists with other diseases, treatment for that disease must also be carried out along with antimony and potassium tartrate administration. For example, Gruzhit[33] found Vincent's organisms associated with granuloma lesions in one instance and diphtheroid bacilli in another. Healing was slow with tartar emetic until the associated diseases were gotten rid of; the first by salvarsan, and the second by administration of diphtheria antitoxin. Cole, Miskjian and Rauschkolb[31] found Vincent's infection in ulcers of the labia. After the Vincent's infection was cleared the ulcer did not heal, and by further study Donovan bodies were obtained and treatment administered with good results. Syphilis is more commonly found associated than is any other disease, and where this is true, as has been reported by a large number of authors, more rapid healing is obtained if antisyphilitic treatment is administered along with antimony and potassium tartrate intravenously. Some workers have diluted the tartar emetic solution with large quantities of saline solution, but usually the 1 per cent solution is either given direct from sterile ampules, or is diluted with 3 to 5 c.c. of sterile saline solution. The dose is usually 2 c.c. of 1 per cent solution, increased 2 c.c. for each succeeding dose until 10 c.c. of the 1 per cent is reached. Most authors at the present time do not give more than 10 or 12 c.c. at one time. The doses may be given daily, every other day, or three times a week, depending on the amount of improvement obtained and the extent of the pathology noted. McIntosh[43] prefers very small doses given twice daily.

Usually after the first few injections the secretions diminish rapidly, the lesions take on a healthy appearance and epidermization begins, and in a few weeks is complete. Because of the likelihood of recurrence in a moderate percentage, a final series of injections of antimony and potassium tartrate solution is advocated by Cuthbert[22], Johns and Gage[40] and many other authors. In a recent personal communication, McIntosh[43] informed the author that his experimental lesion, obtained by a tissue transplant containing Donovan bodies, which had been supposedly cured by administration of antimony and potassium tartrate, returned one year later and showed a recurrent lesion of granuloma venereum at the original site of the experimental lesion.

Conclusion: Granuloma venereum is a distinct clinical entity, and while it usually begins in the genital region, it may affect the skin and mucous membrane and other portions of the body. It is especially prone to attack the mucous membrane of the mouth, and although it is rare in the pharynx and larynx, when these anatomical structures are attacked, marked tissue destruction may be expected and the true diagnosis is especially difficult because of the rarity of the disease in these structures.

BIBLIOGRAPHY.

1. Fernandez, Antonio A.: Semana Med., Buenos Aires, 1923, xxx, Pt. 2, 1113-1126.
2. de Sousa Aramjo Heraclides Cesar: Brazil Med., 1915, xxix, 201.
3. Schochet, S. S.: Surg. Gynec. Obst., 1924, xxxviii, 759-767.
4. Goodman, Herman: Jour. A. M. A., 1922, lxxix, 815-819.
5. McLeod, K.: *Indian Med. Gaz.*, 1882, xvii, 120-122.
6. Conyers and Daniels: Brit. Guiana M. Ann., 1896, viii, 13.
7. Galloway, James: *Brit. Jour. Dermat.*, 1897, ix, 133.
8. Dempwolff: Arch. f. Schiffs-u. Tropen-Hyg., 1898, ii, 134-166.
9. Maitland: *Brit. Med. Jour.*, 1906, i, 1463.
10. Donovan, C.: *Indian Med. Gaz.*, 1905, xl, 411-414.
11. MacLeod, J. M. H.: *Brit. Jour. Dermat.*, 1907, xix, 73-75.
12. Sequifera, J. H.: Proc. Roy. Soc. Med., 1908, Sect. Dermat, i, Pt. i, 92.
13. Row, R.: *Quar. Jour. Mic. Soc.*, 1909, liii, 747-754.
14. Jackson, E. S.: *Austr. Med. Gaz.*, 1911, cxxx, 133-136.
15. Aragao and Vianna: Brazil Med., 1912, xxvi, 283-285; Brazil Med., 1913, xxvii, 41-42.
16. Grindon, Joseph: *Jour. Cut. Dis.*, 1913, xxxi, 236-240.
17. Symmers and Frost: *Jour. A. M. A.*, 1920, lxxiv, 1304-1306.
18. Beatti, M.: Semana Med., Buenos Aires, 1916, xxiii, 281-282.
19. Posados and Roffo: Prensa. Med., Argentina, 1917-18, iv, 323-326.
20. Silva, F.: Rev. franc. de dermat. et de Venereol, 1926, ii, 480-495.
21. Clement, R.: Presse Med., 1926, xxxiv, 1429-1431.
22. Cuthbert, C. F.: Proc. Roy. Med. Soc., 1919-20, Sect. Dermat., xiii, 32-34.
23. Bonne, C.: *Jour. Trop. Med.*, 1917, xx, 109.
24. Cumming, H. L.: *Brit. Med. Jour.*, 1920, ii, 775.
25. Driver, J. R.: *Jour. A. M. A.*, 1922, lxxix, 867-879.
26. Sabella and Wise: Policlinico (sez. med), 1913, xix, 235-240.
27. de Matta, A. A.: Gaceta Medica, Caracas, Venezuela, 1916.

28. Darwent, E. N.: Trans. Soc. Trop. Med. and Hyg., 1916-17, x, 198.
29. Winfield, J. M.: Med. Rec., 1922, ci, 57-60.
30. Choyce, C. C.: Proc. Roy. Soc. Med., 1912-13, Sect. Dermat., vi, 87.
31. Cole, Miskjian and Rauschkolb: Dermat. Ztschr., 1928, liii, 127-143.
32. Pijper, A.: S. African Med. Rec., 1918, xvi, 20.
33. Gruzhit, O. M.: *Amer. Jour. Trop. Med.*, 1923, iii, 289-295.
34. Scott, J. F.: Northwest Med., 1926, xxv, 40-41.
35. Fraser, A. R.: *Brit. Jour. Dermat.*, 1925, xxxvii, 14-29.
36. Wolff, Walter: Med. Klin., 1927.
37. Horwitz, J. L.: *Med Jour. and Rec.*, 1924, cxix, 199.
38. Ross and Kaupp: Northwest Med., 1927, xxvi, 543-549; Personal Communication, Oct., 1929.
39. Thierfelder and Thillot: Munchen. med. Wchnschr., 1926, lxxiii, No. 14, 561-563.
40. Johns and Gage: Internat. Clin., 1924, iv, No. 34, 15-20.
41. Gage, I. M.: Arch. Dermat. and Syph., 1923, vii, 303-325.
42. Low and Newhan: *Brit. Med. Jour.*, 1916, ii, 387-389.
43. McIntosh, J. A.: *Jour. A. M. A.*, 1926, lxxxvii, 996-1001; Personal Communication, Oct., 1929.
44. McRae, J. C.: *Jour. A. M. A.*, 1925, lxxxiv, 512.
45. Purcell, H. M.: Southwestern Med., 1926, x, 309-310.
46. Applehaus, W. E.: *Ken. Med. Jour.*, 1928, xxvi, 192-195.
47. Bergstrand, Hilding: Acta oto-laryng., 1928, xii, 461-467.
48. Randall, Small and Belk: Surg. Gynec. Obst., 1922, xxxiv, 717-739.
49. Knowles: Diseases of the Skin, 1914, 10.
50. Fox, Howard: *Jour. A. M. A.*, 1926, lxxxvii, No. 22, 1785-1790.
51. Weinberg, M.: *Jour. Urol.*, 1923, ix, 505-517.
52. Flu, P. C.: Arch. f. Schiffs-u. Tropen-Hyg., 1911, ix, 87.
53. Castellani, A.: Proc. Roy. Soc. Med., 1928, xxi, 1-16.
54. Rice, F. W.: *Amer. Jour. Obst. and Gynec.*, 1927, xiv, 249-250.
55. Willmott, C. B.: *South. Med. Jour.*, 1928, xxi, 872-875.
56. Sidlik, D. M.: Arch. Dermat. and Syph., 1927, xv, 703-708.
57. Taussig, F. J.: *Amer. Jour. Obst. and Gynec.*, 1923 vi, 411-414 (discussion, 495-500).
58. Hunter, R. J.: Trans. College Phys., Sect. Otol. and Laryngol., 1923, xlv, 455-462.
59. Lynch, K. M.: *South. Med. Jour.*, 1922, xv, 688,692; *South. Med. Jour.*, 1919, xiii, 246; *Jour. A. M. A.*, 1921, lxxvii, 925.
60. Sargent, J. C.: *Jour. A. M. A.*, 1927, lxxxviii, 1394-1395.
61. Destefano and Vaccarezza: Semana med., 1923, 229; Semana med., 1925, 1013.
62. Figi and Thompson: *Jour. A. M. A.*, 1928, xci, 637-643.
63. Friedlander, D.: *Calif. State Jour. Med.*, 1913 xi, 162.
64. McLean, J. B.: *Australian Med. Gaz.*, 1911, xxx, 137-139.
65. Carty, J. R.: Radiology, 1927, ix, 334-335.
66. Goldzieher and Peck: Virchows Arch. f. path. Anat., 1926, cclix, 795-814.
67. Randall, A.: *Jour. Urol.*, 1923, ix, 491-504.
68. Shattuck and Little: *Amer. Jour. Trop. Med.*, 1926, vi, 307- 317.

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THE INCIDENCE OF VINCENT'S ANGINA IN THE STUDY OF FIFTY UNSELECTED CASES.*†

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In our routine work we have been finding patients with clear-cut clinical symptoms of Vincent's angina, some of which are reported positive bacteriologically, others of which are reported negative. We also find patients with symptoms which we are unable to account for clinically, but which show Vincent's by laboratory procedure. Some of these patients clear up on treatment directed at Vincent's. In mouth and throat smears in instances in which certain symptoms prevail but Vincent's was not suspected, Vincent's was shown bacteriologically. In, we believe, as many instances where Vincent's was suspected, it could not be demonstrated by laboratory procedure. Such procedure aroused a certain curiosity on our part and we decided to examine a series of smears for a basis for our conclusions. Fifty unselected specimens covering the hospital staff, nurses and patients, and patients as they appeared at the offices, whether for eye or ear, nose and throat symptoms, were made. We classified patients as to mouth symptoms or none, noting any symptoms that any patients might have attributable to infection in teeth, gums, tonsils or pharynx.

Twenty-four specimens, or 48 per cent, were diagnosed in the laboratory as Vincent's angina with gentian violet stain. Seventeen specimens were diagnosed positive Vincent's by the Gram method. Each Gram stain was corroborated by the gentian stain, but seven gentian stains were not corroborated by Gram stains. Literature states that the Gram method is variable on the organisms of Vincent's angina. Of the 24 positive specimens, six had symptoms of bleeding gums, sore throat, temporal pain, sore hard palate or sore teeth. Nine patients who had symptoms from which one would expect to find organisms, in view of the fact of finding them in like specimens, were negative.

Of 18 positive specimens with no symptoms, five were connected with the hospital and one was a patient, age 3 years, who has been a resident here for over a year. Three connected with the hospital

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were not positive. One patient, a 2-year-old baby, was hospitalized because of laryngeal diphtheria. Smears from the tracheotomy tube, teeth and throat were negative for Vincent's and, incidentally, were negative for diphtheria bacilli on both direct smear and culture. The patient, however, responded promptly to antidiphtheritic measures. One patient, without any symptoms whatever but who was reported positive, came to the office three or four days later with the complaint of pain in and around the incisor teeth. Another smear was positive and registered the same bacteriologically as the previous smear. This condition subsided promptly on treatment directed at Vincent's. One patient with peritonsillar abscess was negative. One patient who had had a tonsillectomy three weeks before and complained of dysphagia as the only symptom, tonsil pillars and fossae were clean, was found positive for Vincent's. The Wassermann reaction in this patient was also positive. Whether syphilis or Vincent's angina was responsible for the pain could not be ascertained because the patient did not return to the office.

Patients with symptoms of Vincent's showed the same bacteriology as positive smears from patients with no symptoms. Our laboratory diagnosis was made by comparing the slides of our series with slides from patients with clear-cut clinical symptoms.

By the gentian method eight organisms were found; large bacilli, fusiform bacilli, spirilli, staphylococci, streptococci, diplococci, spore formers and bipolar bacilli. Five Gram positive organisms were found: staphylococci, streptococci, bacilli, diplococci and fusiform bacilli. Three Gram negative organisms were found: spirilli, bacilli and staphylococci.

In literature, Vincent's angina is variously named diphtheroid angina, ulceromembranous chancriform or ulcerative lacunar tonsillitis, trench mouth and putrid sore mouth. The bacteriology is described as a fusiform bacillus, associated with a spirillum. There are differences of opinion on staining properties.

Classical textbook symptoms are those of ulceration of the gums and tonsils, occasionally in the larynx and pharynx, fever, local adenitis, putrid breath, increased salivation, dysphagia. One author states that Vincent's is contagious, that one suffering from it should be isolated. Another author states that the infectivity has not been proved.

Treatment by applications of tincture of iodin, copper sulphate, silver nitrate, sodium perborate, salvarsan or other arsenicals, locally or intravenously, and alkaline mouth washes has been described. It is our opinion that cleansing mouth washes as sodium perborate and

hydrogen peroxid will suffice in most cases. Aggravated or long-standing cases may react promptly upon administration of salvarsan locally or intravenously.

Summary: The fact that five of eight hospital specimens were diagnosed in the laboratory as Vincent's would bear weight toward the infectiousness of Vincent's.

Bacteriological diagnosis alone is futile in the selection of cases for treatment.

Treating only those cases presenting classical symptoms, as described in textbooks, seems to us to be overlooking the greatest number of cases of Vincent's angina. Not one of our 50 cases had textbook symptoms, but the symptoms that presented clear up on treatment directed at Vincent's angina.

ANALYSIS OF CASES OF LARYNGEAL CARCINOMA SEEN SINCE OCTOBER, 1929.*

DR. JOHN E. MACKENTY, New York.

Laryngectomy *versus* Thyrotomy brings forward the much discussed and yet unsettled question of incipiency. Under what conditions may we be assured, from the location, size and character of a laryngeal cancer, that it is localized and curable by local removal? It is conceded that the growth must be small, superficial and situated in the middle third of one or other vocal cord. The determination of the character, *i. e.*, the degree of malignancy, may be approximately adumbrated by biopsy and, if time permits, by observing the rapidity of the growth. Both of these methods are open to criticism, since the former must tend to squeeze cancer cells into the adjacent lymph channels and the latter must waste valuable time. Yet biopsy is at times inevitable. I can offer no fixed rules for guidance, other than those well known to you. The clinician, facing this vital parting of the ways, can do no better than to rely upon his clinical experience, making his decision on the side of safety. I can think of no more harrowing experience to the doctor or of more abysmal despair

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to the patient than the recurrence of cancer after thyrotomy. In my personal cases, 35 per cent of the thyrotomized patients returned with recurrence. This bitter experience has made me lean towards radicalism in cancer in all situations.

I may err on the radical side as, no doubt, have the thyrotimists erred on the conservative side, but the satisfaction of seeing one's patients alive must condone my error to a considerable extent.

For some unexplained reason, I rarely see the incipient case. Only rarely does a cancer amenable to thyrotomy present itself. Since Oct. 1, 1929, one apparently incipient cancer was sent to me by Dr. Chamberlin, of Cleveland: female, age 30 years, cancer in anterior commissure, involving both cords. It was practically all removed by biopsy done by Dr. Pitkin in Cleveland. The growth proved to be of the worst malignant type. Dr. Chamberlin and Dr. Pitkin, also Dr. Jackson, advised total laryngectomy, on account of situation and degree of malignancy.

Since last October, I have seen 60 cancers of the larynx. The most hopeful of these were operated (10) and will be shown on the screen, omitting Dr. Chamberlin's case mentioned above. This larynx was misplaced and was found too late to be included.

Some might take exception to doing a laryngectomy on Dr. Chamberlin's patient, who had only an early anterior commissure involvement. In my opinion any involvement of the anterior commissure should be treated by laryngectomy.

These pictures show advanced disease and, from that point of view, the most discouraging series I have ever had in so short a period. This presentation of forlorn hopes is, to say the least, pathetic and is an argument for greater vigilance on the part of the physician and earlier submission to examination on the part of the patient.

In this series there have been no deaths or complications. Shortest period of healing, 11 days; longest, 22 days. It may be interesting to note that the direction of growth was more rapid in the forward and downward direction, less in the upward, and still slower in the backward, toward the arytenoids. On account of the extent of the disease, all have been treated, postoperatively, by X-ray (Regaud method).

Summary: Nine larynges of borderline, intrinsic and beginning extrinsic carcinomata were demonstrated. There were altogether 10 laryngectomized patients, only two of whom had frankly intrinsic carcinomata.

111 East 61st Street.

SPOROTHRIX INFECTION INVOLVING THE BONES OF THE FACE.*

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Sporotrichosis is a rather uncommon disease, caused by the invasion of tissue by the sporotrichum, one of the fungi imperfecti. The disease is usually localized in the skin and subcutaneous tissues, although the mucous membranes, musculatures and bones may occasionally be involved. Sporotrichosis of the bones, according to Altschul[1], "belongs to the rarest of diseases." Beurmann and Gougerot[2] state that the tibia is the bone most frequently involved. Although quite a few cases of sporothrix infection of the eye and adnexa have been reported, only 14 references to involvement of the bones of the face were found in the literature. Because of the difficulty of pathological diagnosis it may be that many cases have been overlooked. The histological changes are not very characteristic and the demononstration of the organism in the tissues is extremely difficult. In the case I shall report, the first pathological diagnosis was "giant cell sarcoma." Sporothrix grows well on glucose agar, however, if sufficient time is given, so this may be the diagnostic method of choice. Two different strains are mentioned in the literature. Sporothrix Schenkii and Sporothrix Beumanni. There is some doubt as to whether this distinction is justifiable, however, as there are good grounds for considering them as identical.

Fage[3], in 1908, reported a case of sporothrichosis with multiple gummatous lesions on various parts of the body, one involving the bony wall of the orbit. In 1909, Bonnet[4] reported a case with multiple lesions, one involving the frontal bone. The same year, Beurmann and Gougerot[5] reported a case in a man, age 65 years, with multiple subcutaneous gummata and abscesses. The nasal bone was involved. This case resulted fatally. In Velter's[6] case (1910), the infection seemed to originate in the wall of the orbit and later extended to the temporomalar region. Morax's[7] case (1910) showed involvement of the inner wall of the orbit. Pautrier and Richou[8] (1911) reported a case of sporotrichosis of the nose, in which the lesion invaded the vomer, causing a perforation of the septum. This subsided under treatment with potassium iodid, together with local

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applications of iodin. In one of the cases reported by Lapersonne[9], in 1912, there was considerable destruction of the external table of the frontal bone.

D'Agata[10], in 1916, reported a case in which there were two bone lesions, one involving the tibia, the other on the upper jaw. Both lesions were of the nature of an osteoperiostitis. Sporotrichum Beurmanni was cultured from necrotic material removed from the jaw lesion.

Aloin and Vallin[11], in 1920, reported a case in which the first lesion noted was in the right submaxillary region. This broke down with the formation of a fistula. Later there was a fluctuant area of

Fig. 1. X-ray before operation showing destruction of malar and outer angle of orbit.

swelling over the frontal region. At operation it was found that the frontal bone was necrosed and the dura covered with fungoid growths. Under treatment with potassium iodid the lesions healed. In 1921, Schloffer[12] reported a case in which there were swellings on the jaw and other bones of the skull. Sporotrichum Beurmanni was isolated from the pus from these areas and also obtained from the blood. In 1922, Warfield[13] reported a case with multiple gummata due to sporothrix infection. One lesion had eroded the frontal bone.

In Gifford's[14] case, reported in 1922, in which the infection originated in the lacrimal sac, there was extension into the ethmoidal cells. After intranasal exenteration, followed by treatment with potassium

iodid and local applications of tincture of iodin the lesion slowly healed. Sporotrichum Schenckii was cultured from this case.

In 1923, Mason and Frost[15] reported a case of sporotrichosis in a woman age 53 years, a native of California. The primary lesion was on the thigh. Eleven months later, a firm, painless nodule developed on the forehead, apparently arising from the periostium of the frontal bone. Sporothrix was cultured from this lesion. In spite of treatment the case resulted fatally.

In 1926, Altschul[1] reported a case in a woman age 63 years, in which the first symptom noted was pain in the lower jaw. A hard, immovable tumor slowly developed on the left mandible. The Roent-

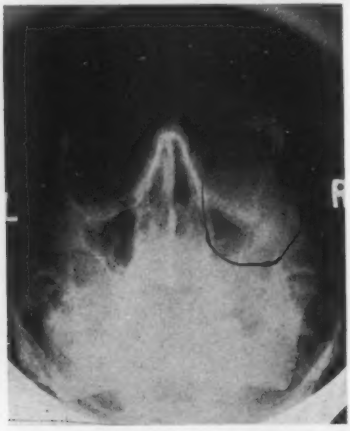

Fig. 2. Showing bone removed at operation.

genogram showed several sharply outlined areas of rarefaction, resembling a multilocular bone cyst, in the jaw. A portion of the tumor was removed and showed a fibrous osteomyelitis. Sporothrix was cultured from this tissue. Subsequently similar tumors developed on the forehead and various parts of the cranium. A Roentgenogram of the skull showed it to be "studded with large and small areas of rarefaction." Potassium iodid and Roentgen-ray therapy were given without effect. Death was due to pneumonia. Necropsy showed defects in the frontal and parietal bones, through which extruded grayish-red material. There was a swelling in the left mandible with a small, oozing fistula. There were large plaques of tumor-like growths on the dura mater, which had partially eroded the bone of the skull cap. There were also bone lesions in other parts of the

body. The author noted the similarity of the Roentgen findings in this case to those of multiple myeloma.

Case Report: G. G., age 32 years, of French-Canadian parentage and by occupation a telephone linesman, had been under the care of Dr. H. F. Hill for the past three years, consulting him first for a small growth on his right upper eyelid. This had been noted 12 years before, at which time it was about the size and shape of the head of a match. This had slowly increased in size until it was about 6 m.m. in diameter. There were no other symptoms. One year previously this had been opened and curetted by his family physician. Wassermann test was negative. Removal of the growth had been

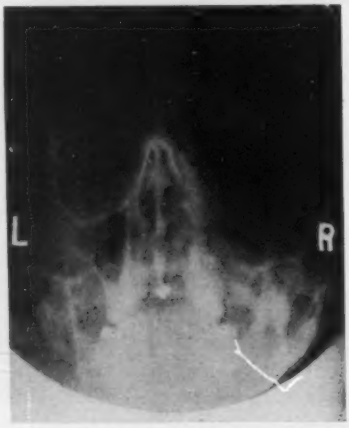

Fig. 3. X-ray after operation.

attempted by Dr. H. F. Hill. The first pathological report obtained was "giant cell sarcoma." Another pathologist gave the report of "giant cell inflammatory tissue from a fungus growth." This was confirmed by Dr. F. A. Verhoeff as sporothrix. Subsequently the growth had extended laterally beneath the skin, infiltrating the outer canthus with the formation of a fistula. Large doses of potassium iodid and Roentgen-ray treatments had been given without effect. Salvarsan had been used intravenously. Locally, iodin and gentian violet had been employed. From time to time, the larger lesions had been removed. At the last removal, six months before, the periosteum at the outer edge of the orbit had been found involved. During the previous year-and-a-half, enlarged lymph nodes had been removed from both inguinal regions and the right side of his neck by

his family physician. Some of this material had been examined but no organisms were found.

When the patient was referred to me there was marked chemosis and exophthalmos, with an indurated nodular swelling extending downward from the outer edge of the orbit and involving the malar bone. He had been suffering constant intense pain for the previous three weeks, the first discomfort he had had. Roentgen examination showed "partial destruction of the right malar bone and of the external angular process of the frontal bone, with areas of rarefaction. Antrum cloudy." Radical surgery seemed to offer the only hope of relief.

Under rectal anesthesia, after removal of the eye by Dr. H. F. Hill, an attempt was made to exenterate the entire mass. This was

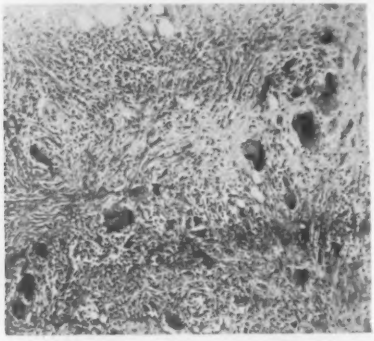

Fig. 4. Section stained with hematoxylin and eosin showing giant cells and fibrosis.

done by a combination of Rongeur forceps on the bone and surgical diathermy on the soft parts, going well around the involved areas. The lower eyelid and the major portion of the upper were sacrificed. As the growth had apparently burrowed beneath the skin and did not involve it lower down, a flap with its base on the nose was thrown back, and the underlying structures removed. The orbit was completely exenterated, its outer wall removed well out to the zygomatic arch and the floor taken away, exposing the interior of the antrum and removing its lateral wall. The bone had the peculiar appearance and feel of "rotten ice." Where the growth had invaded the soft parts it had a dense fibrous appearance. The membrane lining of the antrum was apparently not involved, nor had the growth extended into the ethmoid region. After the removal was completed, the skin

flap was sutured across the lower portion of the opening and it was lightly packed with iodoform gauze.

He reacted well from the operation and experienced almost immediate relief from the previously constant pain. Strange to say, he had little difficulty in chewing, such as one might expect from the removal of the anterior anchorage of his zygoma. After the packing was removed, daily exposure to the Kromayer lamp was given. Under this treatment, together with the applications of gentian violet, healing slowly took place. The orbit gradually filled in and became epidermatized. Cultures taken at various times were negative for sporothrix. The outlook appeared quite optimistic, until four months afterwards, when a nodular swelling was noted in the canine fossa

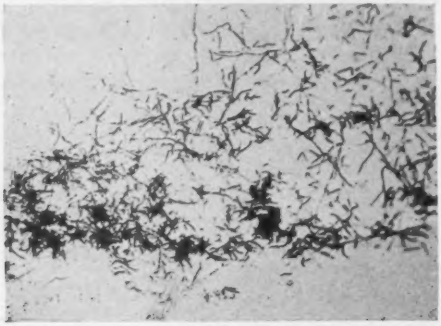

Fig. 5. Section of culture media showing sporothrix growing on glucose agar, stained with Verhoeff's modified Gram stain.

extending backwards. At this time he had a return of the pain in this region.

Under local anesthesia, an incision was made in the canine fossa, exposing a dense, white, fibrous mass, similar to that removed at previous operation. This was found to be much larger than expected and to extend backward almost to the ramus of the mandible. In order to dissect this free it was necessary to reopen the old incision in the cheek, fully exposing the site of the previous operation. Because of this we were able to check up on the condition of the old operative field. In the upper portion, where the floor of the orbit had been removed, there was no sign of recurrence. There was still no evidence of involvement of the antral lining membrane. Infected bone was found at the outer-inferior angle of the antrum in what remained of the lateral wall. As a result of the previous operation the quadratus labii superiores, canines and zygomaticus muscles had

been practically obliterated. At this time the process had involved the superficial portion of the masseter. The growth exhibited a peculiar fibrous characteristic where it had invaded muscle, showing distinct lines of cleavage, something like vegetable root fibres. There was almost no bleeding. Cutting into the growth was almost like cutting raw potato. Removal was very difficult and resulted in tearing into Steno's duct, so that there was some free flow of parotid secretion into the wound subsequently. The canine fossa incision was closed but the upper one kept open for better observation of the field. This was lightly packed with iodoform gauze, which was removed after 48 hours. Exposure to the Kromayer lamp was resumed and has been continued since at intervals of about three days.

Since this operation there has been freedom from the pain. He is feeling well, and is out and doing about as he likes. Recent Roentgenograms of his skull showed no further bone involvement. We

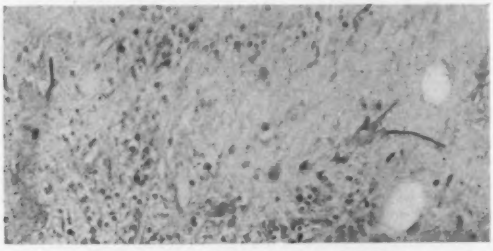

Fig. 6. Section stained with Verhoeff's modified Gram stain showing sporothrix in tissue.

were especially on the lookout for areas of rarefaction in the skull cap as noted by Altschul. The possibilities of metastatic involvement of the lungs, and,—bearing in mind the enlargement of the inguinal nodes — of invasion of the pelvis, warrant careful consideration. Roentgen examination of these areas have so far been perfectly negative. One cannot help but be very pessimistic regarding the prognosis. The continued slow progression of the process, despite therapy and surgery, makes one dubious of the possibility of cure. Had radical surgery been applied earlier, when the lesion was apparently regional and circumscribed, there would have been better chance of complete eradication.

It has been puzzling to account for the etiology of this infection. According to Gifford[16], sporotrichosis most commonly involves the hand following some superficial wound or abrasion. This patient at no time has had any lesion on his hands, or elsewhere superficially

on his body, except the eyelid. In inquiring into his occupation, it seems that his work as a linesman was on cross-country telephone lines. During his noon-day lunch hour he was in the habit of taking naps lying face downward on the ground. It might be possible that he got his primary infection on his eyelid in this manner. However, this disease is extremely rare in New England, although not so uncommon in the Mississippi basin, and this man has always lived in Maine.

Pathology: The pathological work on this case was done by Dr. F. A. Verhoeff, whose isolation of the sporotrichum earlier in the course of the disease had established the diagnosis. His report is as follows:

"All the tissues submitted for examination, including the bone tissue, except the lymph gland, show the same inflammatory process. The normal tissue has been pervaded with granulation tissue which has gone on to the formation of dense fibrous tissue. The tissue contains large numbers of foreign body giant cells, but no tubercles resembling tuberculosis or syphilis. Some of the tissue shows considerable infiltration with lymphocytes; some, only moderate infiltration with these cells. By ordinary methods of staining, no organisms can be found in the tissues, but Verhoeff's modified Gram stain shows filamentous organisms scattered through the tissues, some of them of great length. Some of them are surrounded by giant cells, others are not. The lymph gland shows the lymph sinuses distended with epitheloid cells. No organisms can be found in the lymph gland.

"Smears from the pus from the lesion in the lids shows sporothrix filaments in considerable numbers. A culture taken on glucose agar showed the organism in four days. Transplants from the original growth on glucose agar and Sabaraud's media failed to grow. Pieces of the original culture media were fixed in formalin, sectioned and stained in Verhoeff's modified Gram stain. These show the organisms growing down into the medium in characteristic fashion."

Potassium iodid has been considered as almost a specific in these cases. Gifford[16] says that in large doses it nearly always brings about a prompt recovery. However, in this case prolonged treatment with potassium iodid, to the point of toleration, for four years, has been useless. Several of the cases with bone involvement, reported in the literature, also have shown no improvement and have resulted fatally. It would seem probable that any effect that potassium iodid has in these cases is produced by breaking down the fibrosis, as in tertiary lues, and thus allowing the natural defensive powers of the body, or such other therapeutic agents as may be employed, to act

upon the organism; rather than by any specific reaction for the sporotrichum, inherent in the drug itself.

Summary: A case of sporotrichosis is reported; of 12 years' duration, beginning with a small circumscribed lesion on the eyelid. This was apparently activated by curettage into a slow, but persistent progression, finally involving the bones of the face. Iodin, salvarsan and Roentgen-ray therapy have been of no avail. There seems to have been a predilection primarily towards bone and later towards muscular involvement, with little tendency to invasion of the skin or mucous membranes. Pain was a late symptom and apparently due to pressure. Radical surgery, at the time instituted, at least from present indications, has only been palliative.

Conclusions: From the meager material available it is useless to attempt to draw conclusions. However, the following points seem to present themselves for consideration:

1. Sporotrichosis of the bones is extremely rare.

2. Its diagnosis is difficult and may be overlooked, or confused with neoplastic disease. Cultural diagnosis is easier.

3. Its prognosis is dubious. Potassium iodid therapy is not always successful.

4. Incomplete surgery is inadvisable. If surgery is undertaken it should be radical and done if possible when the lesion is circumscribed.

BIBLIOGRAPHY.

1. ALTSCHUL, W.: Similarity of the Roentgen Findings in Multiple Myeloma and in Sporothrichosis. *Amer. Jour. Roentgenol.*, 15:224, 1926.
2. BEURMANN AND GOUGEROT: Les Sporotrichoses. Paris, 1912.
3. FAGE, A.: Sur un cas de sporotrichoses. Progres Méd., 24:240, 1908.
4. BONNET, L. M.: Sporotrichose à localisations osseuses et museulaire. Lyon chir., 1:683, 1908-09; 2:515, 1909.
5. BEURMANN AND GOUGEROT: Sporotrichose cachectisante mortelle. Bull. et mém. Soc. méd. d. hop. de Paris, 27:1046, 1909.
6. VELTER, E.: Un cas de sporotrichose orbito-palpébrale primitive. Ann. d'ocul., 144:72, 1910.
7. MORAX, V.: Sporotrichose der Orbita. Ber. ü. d. Versammlung d. ophth. Gesellsch., 36:331, 1910.
8. PAUTRIER AND RICHOU: Sporotrichose du nez. Bull. d'oto-laryngol., 15: 54, 1911.
9. LAPERSONNE, F. DE: Sporotrichose oculaire. Presse méd., 20:93. 1912.
10. D'AGATA, G.: Sporotrichosia ossea. Policlin. sez. chir., 23:1, 1916.
11. ALOIN, H., AND VALLIN, H. G.: Ostéite perforante du frontale par sporotrichose. Lyon méd., 129:859, 1920.
12. SCHLOFFER: Ein Fall von extra-Cutaner Sporotrichose. München. med. Wchnschr., 68:503, 1921.
13. WARFIELD, L. M:. A Case of Disseminated Gummatous Sporotrichosis. Trans. Assn. Amer. Physicians, 36:5, 1921; also in *Amer. Jour. Med. Sci.*, 164: 72, 1922.
14. GIFFORD, S. R.: Ocular Sporotrichosis. Arch. Ophthal., 51:540, 1922.
15. MASON, V. R., AND FROST, K. P.: Report of a Case of Sporotrichosis. *Calif. State Jour. Med.*, 21:471, 1923.
16. GIFFORD, S. R.: Diseases of the Eye and Adnexa Due to Fungi and the Higher Bacteria. Arch. of Ophthal., 57:224, May, 1928.

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FURTHER STUDIES IN NON-SUPPURATIVE DISEASES OF THE NASAL SINUSES: THE RELATIONSHIP TO SPECIFIC PROTEIN SENSITIVITY.*

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In my article on the neurologic aspects of nasal sinus infections, I pointed our that a chronically thickened sinus mucosa is the source of widespread disturbances along the pathways of the facial nerve, the trigeminal nerve and the vegetative nervous system. I also pointed out that surgical intervention on the sinuses in these cases is both incomplete and essentially ineffective, and has given rise to an almost unwarranted amount of skepticism regarding the value of sinus surgery. In no type of case has surgery of the sinuses been more unhappy than in the cases of allergy and protein sensitivity. The amount of failure in these cases has been so great that many of those treating allergy cases advise against surgery even in cases that are distinctly suppurative and would benefit greatly by adequate sinus surgery. The blocking of the nerve pathways by injection of the sphenopalatine ganglion frees the patient from the irritating impulses arising from the diseased areas in the nasal sinuses and by its influence on the vasomotor supply diminishes the secretion and causes shrinking of the engorged mucosa. The injection of the sphenopalatine ganglion thus obviates surgery in the nonsuppurative sinus cases.

The problem becomes much more complicated when associated with chronic asthma and chest pathology. In these cases injection of the nasal ganglion gives only partial benefit.

It has been practically generally accepted that the treatment of cases showing a definite sensitiveness to a specific protein should be desensitization by the injection of the specific protein. It is therefore of interest to report the following case of specific sensitiveness to fish and meat associated with nonsuppurative sinusitis relieved directly by injection of the sphenopalatine ganglion.

Case Report: Miss A. R., nurse, complained of sneezing, profuse watery nasal discharge and mild asthmatic attacks ever since an attack of influenza in 1918. These attacks come on only after eating fish or meats. She has therefore been obliged to refrain rigidly from these foods. In 1927, she underwent a careful study at one of

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the national sanitariums. They very kindly supplied me with the reports of their examination. The tests made were as follows: *Meat Group:* Beef, lamb, mutton, pork, veal. *Special:* Milk, whole egg. *Fish Group:* Bluefish feret, pike, salmon, shad, sole. *Fowl Group:* Chicken, duck, goose, guinea hen, squab, turkey. *Special:* White potato. *Vegetables:* Asparagus, celery, eggplant, garlic, onion, radish. *Special:* Tomatoes, nut groups: Almond, chestnut, peanut, pecan. *Special:* Wheat, oatmeal, orange. *Results:* Strong reaction to fish, XXXXX; meat groups, XX; chicken, XX.

Summary: Patient's reaction to fish is so strong that we believe it must be a factor in her bronchitis or asthmatic attacks. We have advised her to strictly avoid fish and to be extremely moderate in the use of meat.

In January, 1930, she again returned to the same sanitarium for further study.

A summary of their history record shows the following complaints: *1.* Headache. *2.* Rheumatic pains. *3.* Shoulder pains. *4.* Dizziness. *5.* Asthma. *6.* Sneezing. *7.* Seasonal hay fever. *8.* Sensitivity to fish, etc. *9.* Urticaria. *10.* Spastic colon.

Their X-ray report was definite with marked increase in density of both maxillaries, particularly the right, with possible increased density of ethmoids. The comment was as follows: This patient needs a bilateral Caldwell-Luc operation, but prior to this a submucous resection. The patient was, however, advised to go to Florida till the weather became warm.

Here was a case that would ordinarily be subjected to stripping of the lining membrane of the antra with a possible questionable result. Study of the X-rays revealed a distinctly thickened mucosa throughout the sinuses. I proceeded to inject both nasal ganglia. Although for the last three months practically any protein food would induce sneezing, rhinorrhea, itching of the eyes and tearing, nevertheless she got no such attacks after the injection and has been able to eat all foods indiscriminately for the last two weeks, without suffering any discomfort whatever.

Although this is an isolated case, nevertheless I feel that the detailed report of a carefully studied case is enough to give us a groundwork for the investigation of the relationship between food allergens and nasal neurology.

It also points to a distinct method of affording immediate relief of both nasal etiologic factors and a symptomatic relief in a speedier and easier way than protein desensitization.

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HEADACHES OF NASAL ORIGIN.*

DR. LEO J. HOMBACH, Council Bluffs, Iowa.

A large percentage of people who come to consult the rhinologist mention headache as one of their chief complaints. It is probably the most universally distributed symptom of mankind, and there are few diseases going their courses which may not have it as a symptom.[1] Some of these people suffer almost constantly and look ill, run down and toxemic; others have periodic attacks of violent headache, with itervals of freedom; some suffer in silence in so far as medical advice or treatment is concerned; others seek remedies everywhere, many falling into the hands of the cultists and unscrupulous charlatan. Those who do seek medical attention will undoubtedly be investigated for bowel trouble, errors of refraction, foci of infection, etc., but in comparatively few will the nose be examined. One often suspects that the specialist, dealing in his limited field, sincerely believes that most headaches result from disturbances in his specialty. However, aside from that, I would suggest that the nose be examined for any possible source of trouble, other possibilities having proven negative, before calling it a case of "nervous headache", "toxemic headache", etc.

Pain[2] may be regarded as a reaction of the organism in part or as a whole, to harmful influences giving a warning in consciousness that some activity prejudicial to the health of the tissues is operative. The sensations of pain are transmitted to the brain centers through the afferent nerves, and is referred to the origin of the nerve fibre, whether the irritant is applied at the periphery or to any part of the nerve in its course to the brain. The trigeminus is the chief sensory nerve of the face and nose, and through its ramifications are conveyed many of the manifestations of common sensation in health; and the sensation of pain in headache or neuralgia when its protoplastic elements are disturbed by disease of the surrounding tissue.

The[3] sensory branches of the trigeminus are made up of fibres which spring from the cell bodies of the Gasserian ganglion and pass to the nose by way of the ophthalmic and superior maxillary branches. That is, the peripheral fibres of the neurons progress from

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the Gasserian ganglion to the nasal mucosa by way of these branches and rise free between the epithelial cells of the mucosa. The proximal fibres proceed from the Gasserian ganglion to the sensory nucleus in the pons, where they synapse with secondary descending neurons to the spinal cord, and with ascending intercolating neurons to the sensorium in the cerebral cortex. Sluder[3] states that besides these sensory branches of the trigeminus, there are afferent sympathic and somatic neurons from the nose which carry sensory impulses by way of the great superficial petrosal, the geniculate ganglion, the pars intermedia of the facial, the great deep petrosal by way of the carotid plexus and cervical sympathetic nerves to the spinal cord.

The[2] sphenopalatine ganglion is the anatomical center of distribution of the sensory nerves of the nose. It lies in the sphenomaxillary fossa at the posterior end of the middle turbinate, sometimes immediately under the mucosa, in intimate contact, topographically, with the lateral wall of the nose, sphenoid sinus and certain posterior ethmoid cells. It has three roots, which convey nerve fibres to and from it. The motor root comes from the facial by way of the great superficial petrosal and the sympathetic root from the continuation of the carotid plexus by way of the great deep petrosal. These two nerves join to form the vidian, which in turn connects to the sphenopalatine ganglion. The sensory root consists of the sphenopalatine nerves, two or three in number, connecting the ganglion at its upper border to the maxillary branch of the fifth. The ganglion gives off four branches: the ascending, or orbital; the internal, or nasal; the descending, or palatine; and the pharyngeal; all of which carry secretory and sensory fibres.

Most[3] of the cranial nerves leave the skull through close-fitting bony foramina, as the optic, maxillary and vidian nerves, whose foramina are located in the sphenoid and are subject to the same affectations that beset other parts of the nasal mucosa. The sphenoid sinus sometimes extends to the foramen ovale and to the semilunar ganglion. One can readily see that with recurrent, severe inflammatory attacks in the upper respiratory passage, with changes in the mucous membrane and periosteum of the nose, there will be pressure on the nerves in the foramina or at their peripheral endings in the mucosa, giving rise to pain.

By[4] far the more important condition causing headache of nasal origin is sinus disease, and unsuspected pus held up in one or more of the sinuses is productive of many obscure complaints. The headache in sinus disease is rarely typical as to location, time of appearance or severity, but because of this inconstancy the astute physician will suspect probable sinus involvement.

The diagnosis of the site of the nasal lesion causing pain that is due to acute pathology is rarely difficult. It is in the atypical, chronic conditions, where the location or character of the pain is inconstant, that taxes our patience[3 6]. A very important requisite for recognition of visible signs of disease changes in the nose is an adequate means of illuminating the various recesses in the nasal cavity. The intense white light of the arc lamp advocated by Sluder is quite popular in some sections. The[6] nasopharyngoscope is another indispensable diagnostic aid in many puzzling nasal conditions.

Most[6] men agree with Skillern that transillumination of the sinuses is of questionable value in diagnosing sinus affections. With[4] the advent of lipiodol, the value of the X-ray in diagnosis has greatly increased. The introduction of the oil solution into the sinus, either by direct injection or by the displacement method, now gives the rhinologist information concerning intrasinus changes which he hitherto did not obtain upon the study of the X-ray plates.

Probably[7] the most frequently infected sinus is the maxillary. Here the pain is more atypical than of any of the sinus infections. According to Skillern[6], typical pain from a maxillary infection is directly over the frontal sinus on the same side, due to referred pain from the infraorbital to the supraorbital nerve through their anastomotic branches. Often this frontal headache has been mistaken for frontal sinus infection, and an occasional healthy frontal has been opened. However, the distribution of the pain is usually not as high or as widespread as in a true frontal, and there is generally a feeling of distention or pressure over the antrum, yet it is exceptional to elicit tenderness over the antrum except in severe, acute cases.

The[1] headache produced by frontal sinus infection is the most typical of all the paranasal sinus affections. Here the pain is located over the distribution of the supraorbital nerve. The[8] character of the headache may vary from a dull, heavy feeling in the head, not severe enough to confine him to the house, but enough to greatly interfere with his business, especially in brain work, where concentration is needed, to the most excruciating, unbearable splitting or throbbing headache[9], in which many of the unfortunate sufferers think of suicide to end their agony.

The[10] pain due to ethmoid is not typical of its nerve supply. Here the headache is as a rule located over the parietal region of the skull and is a dull pressure, rather than a pain. In severe suppurative cases the pain may extend over the temporal region and unto the vertex. Often[8] it is located between the eyes, above the root of the

nose, described as if a wedge were prying the nerves apart in the turbinal region.

Next[10] to headache due to frontal sinus infection, the most typical pain is produced by the sphenoid sinus. Usually the pain is in the occiput, having more the character of pressure than of pain. Skillern[9] reports several cases of pain from sphenoid infection over the maxillary sinus. Sometimes, because of the anatomical position of the sphenopalatine ganglion, it becomes involved and there is a reflex pain to the mastoid process temporal region and possibly pain in the shoulder.

Atrophic rhinitis[1] is at times associated with severe headache. The main complaint is of severe generalized headache, due to the sinuses being filled with crusts, as well as the nasal mucous membrane being crusted. Often, when there is no fetor, or mention of it by the patient, the cause is overlooked.

Another[3] cause of headache is a condition named by Sluder, vacuum headache, resulting from closure of the nasofrontal duct so that the air in the sinus is absorbed and a negative pressure is produced, causing congestion of the lining mucous membrane. There is pain in the floor of the sinus internal to and behind the pulley of the superior oblique muscle. The pain is consequently aggravated after the use of the eyes, especially on close work. This condition can be differentiated from asthenopia due to some error of refraction by Ewing's sign, which elicits pain in the floor of the sinus by digital pressure. Ross Skillern[9] says that in his practice he sees very few vacuum headaches. Sam Skillern does not believe there is such a thing as vacuum headache.

Sluder[3] also describes a neuralgia of the anterior ethmoidal nerve, due to pressure on the nerve in its sulcus as it enters the nose. The pain is uniformly in the region of the forehead, limited between the eyebrows, extending above the line of the supraciliary ridge, and below into the nasal bones. Sometimes there may be pain in the orbit and nasal fossae. Often the wearing of glasses is very uncomfortable. The pain is relieved by an application of cocain to the region of the ganglion.

Every rhinologist is familiar with Sluder's sphenopalatine syndrome, explained by the complicated nervous elements of the ganglion, connecting it to the general nervous system through its sympathetic ganglia, etc. Pierre Bonnier[11], in 1907, stated the principal of his theory or doctrine that he called centrotherapy, in which he hoped to relieve pain in the head, and all types of pain generally,

simply by a light cauterization of the mucous membrane of the nose His explanation of the nervous mechanism is somewhat similar to that of Sluder's, in which he (Sluder) explains many of the neurological symptoms originating from the sphenopalatine ganglion. Asuero, of Madrid, took up Bonnier's system of therapy and he has been attracting a great deal of attention on the Continent the past two years because of the wonderful results he claims in all types of cases. Ramon Castroviejo[12], of Fisher's Clinic, during the summer of 1929 ran a series of 500 cases, using Asuero's technique, which consisted of a light cauterization with the actual cautery of the mucous membrane of the middle turbinate and middle and inferior meati. This was followed by massage of the mucous membrane with a special ball-like applicator. Castroviejo's conclusions were that in many cases the pain was relieved, that it was a powerful suggestive method of treatment, but of practically no value in organic disease. He has abandoned this form of treatment entirely.

J. A. Glassbury[13] calls attention to a type of headache which is not at all of nasal origin, but which is often seen by the rhinologist. It is the pituitary headache, due to a disturbance of the secretion of the pituitary gland. I have in mind one case in particular, which came in Dr. Fuller's service at Fisher's Clinic last summer. The patient was a young lady, age 30 years, who had complained of headache for the past four years. She had had five nasal operations by five different doctors, with no relief from the headache. She had the facies of an endocrine dystrophy, so she was put on pituitary extract. After two months she had lost 11 pounds, and was free of her headache for the first time in four years.

To Epitomize: Since the large percentage of the people who come to the rhinologist for advice complain of headache in some form or other, and since we know that headache resulting from sinus infection is rarely typical, it behooves the astute specialist to have an adequate source of illumination which will give him an ample view into various recesses of the nasal fossae, and to become expert in the use of the nasopharyngoscope. When looking for the source of the headache, the specialist should not confine himself to his own limited field, but remember that, even though there may be pathology in the nose, the headache may be due to some other disorder in the organism.

BIBLIOGRAPHY.

1. ATKINSON, E. M.: *Brit. Med. Jour.*, Aug., 1927, pp. 264-266.

2. STAUFFER, FRED: Headaches of Nasal Origin. Eye, Ear, Nose and Throat Monthly, 1922, i, 301.

3. SLUDER: Nasal Neurology and Eye Disorders. The C. V. Mosby Co., 1927.

4. ALDEN, ARTHUR M.: Headache and Neuralgias of Nasal Origin. THE LARYNGOSCOPE, 38:160-164, 1928.

6. SKILLERN, ROSS HALL: Accessory Sinuses of the Nose. J. L. Lippincott Co., 1923.

7. SKILLERN, SAM. R.: Lectures to P.-G. Students, U. of P., 1929-30.

8. SKILLERN, ROSS HALL: Headache—The Nasal Aspect. Bull. N. Y. Acad. of Med., 2nd Ser., 1:133-140, June, 1925.

9. SKILLERN, ROSS HALL: Headaches, Particular Reference to Nasal Origin. P.-G. Seminar, Philadelphia County Med. Soc., April 4, 1930.

10. ETHERIDGE, H. R.: Headaches of Nasal Origin. Vir. Med. Monthly, 55: 127-130, May, 1928.

11. HINOYAR, ADOLPH: Theory of Centrotherapy. Letter to El Sal, April, 1929.

12. CASTRONEIGO, RAMON: Personal Communication.

13. GLASSBURY, J. A.: The Pituitary Headache in Rhinological Practice. *N. Y. Med. Jour. and Med. News*, 115:362, 1922.

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TREATMENT OF LATERAL SINUS THROMBOSIS WITH DICK'S ANTISCARLATINAL SERUM.

DR. MATTHEW S. ERSNER, Philadelphia.

The streptococcus hemolyticus is practically the only organism found in sinus thrombosis. The future prospects are very encouraging because of the results that the Dicks have obtained in the "wholesale" Dick testing for the prevention of scarlet fever.

There is a possibility that the Dick test will become as popular as the present Schick test. Reasoning along these lines, and utilizing our bacteriological knowledge, we feel that the bugbear of mastoiditis may possibly be eliminated in the future. A peculiar coincidence is that upon bacteriological study of suppuration in otitis media and mastoiditis, we found that in about 50 per cent[1] of the cases the streptococcus hemolyticus was isolated. To reiterate, since the streptococcus hemolyticus is the only organism found in sinus thrombosis, we may assume that there may be a close relationship between the scarlatinal streptococcus and the streptococcus found in sinus throm-

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bosis. The Dicks have definitely established the fact that the streptococcus is the cause of scarlet fever. In consulting with Dr. John A. Kolmer, he suggested hypodermic administration of the scarlatinal serum (Dick's serum), the dosage depending upon the age and weight of the patient.

Our results in three cases have been extremely gratifying and we report them as a preliminary presentation. The symptoms in each instance were typical, with temperature, chills and positive blood cultures. Dick's serum in conjunction with intravenous therapy and blood transfusion rapidly brought about a favorable result, with eventual complete recovery. One of these cases had a jugular ligation in addition to the administration of Dick's serum.

Certain precautions are to be taken, chief amongst which are to determine possible anaphylactic sensitivity for the serum, and in those cases found sensitive, begin with 1 c.c. injection and wait one hour before giving additional doses. It is true that at the present time it is difficult to state definitely how the serum acts. There may be the nonspecific protein element producing the results. On the other hand, I venture to say that it would be advisable to seriously consider the specific protein action. The results obtained in the three cases mentioned were so outstanding that scarlatinal serum deserves consideration in the treatment of sinus thrombosis.

REFERENCES.

1. Ersner, Mathew S.: Bacteriological Analysis of Acute Mastoid Disease. *Atlantic Med. Jour.*, June, 1925.

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International Digest of Current Otolaryngology.

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Dr. H. C. Rosenberger, Cleveland.

Castellani, in *The Lancet,* March 22, 1930, writes that he has found that fetor oris of vestibular origin is always associated with the presence of two bacilli, for which he proposes the names bacillus colofoetidus and bacillus alkalofoetidus.

B. Katz, in the *American Review of Tuberculosis,* May, 1930, reports an ingenious method of introducing trichloracetic acid crystals into the larynx in cases of laryngeal tuberculosis. He uses a shallow spoon-shaped container with a sliding cover which can be opened and closed. One or two of the crystals are introduced in the container and locked there. After the larynx is cocainized the closed instrument is introduced directly over the affected part and the container is opened, allowing the acid to cover the affected area. By this technique the healthy surrounding tissue is spared from being burned.

Monroe and Volk, in the May, 1930, issue of the *Journal of Public Health,* report on the effects of 730 tonsillectomies and 741 controls. They analyzed the complaints before and after operation and compared the results with an analysis of complaints in the controlled group. The following conclusions were drawn: Tonsillectomy affords the child much relief from acute upper respiratory infections; it reduces malnutrition and promotes disappearance of enlarged cervical glands. The analysis showed an improvement in 91 per cent of the operated cases. Children with subnormal mentality and retarded school progress showed improvement in 40 per cent of cases after tonsils and adenoids had been removed. The control cases showed no improvement after one year, compared to the 91 per cent improvement in the operated cases.

Cobe, of Philadelphia, in the April, 1930, *Journal of Infectious Diseases,* reports on the incidence of bacteria in 400 tonsil cultures. Staphylococci were predominating organisms, streptococci followed pneumococci in predominance, with hemolytic streptococci the predominating members of the group. Streptococci seemed more common in younger patients. There was a definite seasonal difference in that B influenzae, B mucosus-capsulatus and the diphtheroids being more common in the Spring, while microccus catarrhalis was more prevalent in the Fall.

The Thirty-Fifth Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology will be held at Chicago, Oct. 27-31, 1930, with headquarters at Hotel Sherman. The guest of honor will be Dr. Emile de Grosz, of Budapest, Hungary. Included in the scientific program is a symposium on Diseases of the Nasal Sinuses and Their Relations to Disorders of the Eye. Two lectures on Progress in Oto-Laryngology will be given by Dr. Arthur W. Proetz, St. Louis, and Dr. S. J. Kopetzky, New York. Many other interesting papers are scheduled for the meeting.

In Foreign Letters, *Journal A. M. A.,* June 14, 1930, it is reported that Dr. J. Souza Mendes presented before the Society of Medicine and Surgery of Rio de Janeiro his technique for the drainage of the sphenoidal sinus. The operation is performed under local anesthesia; the first step is the deviation of the nasal septum, the nasal bone being forced to the opposite side; the mucous membrane of the anterior aspect of the sphenoidal bone is separated and the sphenoidal sinuses are transformed into one cavity by resection of the septum between them; the nasal septum is then replaced in the median line and post-operative tamponade is not necessary. The author claims he had good results in three cases with this technique.

W. W. Lewis, St. Paul, in *Minnesota Medicine,* May, 1930, presents an interesting comparison of hyperplastic sinusitis and its parallel, the mucoid ear. The nonsuppurative nasal sinuses may be causing vague general symptoms despite negative findings by the rhinologists. In the same way the mucoid ear may not be recognized. Lewis states that the old-fashioned condition known as "intumescent catarrh," the irregular hay-fever patient and the so-called vasomotor rhinitis patient should be thought of as possible hyperplastic sinusitis cases. He cites cases of low-grade arthritic involvement which cleared up on evacuation of a big glob of mucous. In his treatment of these nasal conditions, the author is extremely conservative. Where he

finds such vague symptom in a narrow, crowded nose, he finds that a high submucous resection has very often been the only procedure necessary to obtain complete relief. He does not favor irrigation of the antrum in cases where the antra are involved, but with very gentle suction over long periods of time he has been successful in evacuating the cavity.

This paper presented by Lewis impresses the abstractor as being one of the clearest, concise and most comprehensive articles that it has been his pleasure to read.

Anderson, of Rochester, Minn., in the *Journal A. M. A.*, June 14, 1930, reviews 400 cases of suppuration in the paranasal sinuses as factors in focal infection. He claims that sinusitis was not a factor in focal infection: teeth, tonsils, prostate and other foci are of more importance. Anderson claims that in a patient with a normal nose, on clinical examination, with a history negative for disease that can be referred to the nose, any exploration which involves mutilation of the nasal membranes is not justified.

Uffenorde, in the Deutsche Med. Woch., May 9, 1930, writes on the diagnostic difficulties in four fatal cases with complicated acute suppuration of the accessory nasal sinuses. In the first case an acute unilateral sinusitis was followed by sepsis, and Uffenorde stresses the importance of the blood picture in making the diagnosis. In another case the patient suddenly developed chills, severe pain in the occiput, in the trigeminus and in the teeth. Necropsy showed that the infection originated in the sphenoid sinus, perforated the posterior orbital cavity and then led to meningitis. Both of the other cases were similarly difficult of diagnosis and the author makes a plea that rhinologists use all methods at their command in making a diagnosis in obscure cases.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

To better carry on its work the American Board of Otolaryngology will increase the certificate fee from $25.00 to $50.00—the new rule becomes effective Jan. 1, 1931.

An examination was held in Detroit, Mich., Monday, June 24, 1930, at which time 78 candidates were examined, of which 65 were passed, one conditioned in pathology, and 12 failed.

The next examination will be held in Chicago, Monday, Oct. 27, 1930. Those interested will please advise the Secretary, Dr. W. P. Wheery, 1500 Medical Arts building, Omaha, Neb.

DR. W. P. WHERRY, *Secy.* DR. H. P. MOSHER, *Pres.*

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF LARYNGOLOGY AND RHINOLOGY.

Meeting of January 22, 1930.

Unusual Rhinolaryngological Conditions and Their Treatment. Dr. Gordon B. New.

Published in full in THE LARYNGOSCOPE, *August, 1930.*

DISCUSSION.

DR. MACKENTY: I wish to thank Dr. New for the pleasure of listening to one of the most instructive and best prepared papers I have ever heard. It would be superfluous for me to say anything on the subject, for I cannot add anything to so large an experience with these rare types of tumors.

I had two cases of atresia of the choanae some years ago, in which I relieved the atresia by removing the posterior part of the septum and placing the choanae further forward in the nose. This gave good breathing and relieved the nasal obstruction.

I was interested in what Dr. New said about one patient, a girl age 16 years, with carcinoma of the larynx. I have reported carcinoma in a girl, age 20 years, with three months' radiation with deep X-ray. The growth was stimulated and made such progress that the entire larynx had to be removed. There was difficulty in closing the neck on account of the radiation. That was two years ago, and there has been no recurrence to date. This year, I removed carcinomatous larynx from a woman, age 30 years; growth was small and located in the anterior commissure but the entire larynx had to come out on account of the extreme malignancy shown in the tissue from biopsy. I have also had two cases of carcinoma of the larynx in women, age 26 years. That makes four cases in females under 30 years of age. One patient, age 29 years, was the youngest man I have seen. We are observing that carcinoma of the larynx is becoming more prevalent in young women; in the old days it was extremely rare under 40 years but now it is not uncommon under 30 years.

I shall not touch upon the other condition Dr. New mentioned, for I cannot add anything to what he has said.

DR. SAMUEL MCCULLAGH: I feel as Dr. MacKenty does, that there can be no discussion of so complete a paper. All I wish to do is to thank Dr. New for his very delightful and instructive talk.

DR. HENRY B. ORTON: I cannot add anything to the paper; it was a wonderful presentation, a fine showing of a mass of unusual recognized clinical material. I also wish to extend my thanks to Dr. New for this presentation.

DR. G. A. WYETH: I should like to ask Dr. New about cases of carcinoma of the larynx in young people. He has had three cases under 22 years of age and I am interested to know if radiation was used before or after operation in these particular cases. If after, one wonders what the result might have been had they been properly radiated before operation. Two of these cases were Grade IV and one Grade III—all anaplastic, embryonal tumors. These less differentiated tumors are highly radiosensitive and should respond to radiation.

DR. GORDON B. NEW (closing): The actinomycosis question is very interesting, and I wanted discussion, for I knew that you have so many western visitors here that you may see cases from time to time. Three or four years ago, we made a geographic study of it. A few cases are seen in New York; but in the West we have many.

Multilocular cyst of the neck, you know, may occur from the clavicle to the jaw, but the most common location is the submaxillary region; the mother brings the child because she thinks the tongue is getting bigger.

The question of radiation of cancer of the larynx: I feel very strongly about it. I don't feel that any cancer of the larynx should be radiated. Fifteen years ago, I went through a number of cases, and though since then certain cases have been cleared up by radiation, I still feel that in cancer of the larynx

the best chance is through surgery. In these cases, because the patient was young, we did a first-stage laryngectomy, and then do a thyrotomy, and later I split the larynx to see if I could get the growth out with conservative surgery. In two of these three cases we did a laryngectomy; in the other, a laryngofissure. Radiation was used after the surgery. In my experience, we do not feel that radium should be used in any cancer of the larynx.

Observations of Agranulocytosis. Dr. Nathan Rosenthal.

Published in full in THE LARYNGOSCOPE, *August, 1930.*

DISCUSSION.

DR. JULIUS A. HAIMAN: This condition of agranulocytic angina is very important, because the general man is called in and finds a patch back of the pharynx or tonsil, or elsewhere, and he then calls in a nose and throat man. These things may at first appear like a Vincent's angina; I was impressed that Dr. Rosenthal did not bring out this point. I was called last year to see the case of a woman, age 48 years, who started with an attack of acute influenza. We examined the sinuses, nose and throat, and there was no nasal patch nor any on the tonsil or larynx. She had marked pain radiating through the ear. This persisted for two or three days. Then the doctor took her blood count, and we were surprised to find the blood count was 600 whites and 6 per cent polys. A diagnosis of agranulocytic angina was made. There was no patch noted in the pharynx or elsewhere. It was interesting to see the lymphoid follicles begin to swell and break down like Vincent's angina. The difference between this and Vincent's angina is that the Vincent's is more of a dirty-grayish patch and the edges are ragged and more punched out. After the diagnosis was made, we immediately started transfusion and neosalvarsan. Men were called in as to the advisability of X-raying the spleen and long bones; finding that to be contraindicated, only small doses of neosalvarsan were given. There was some discussion as to whether the benzol in the neosalvarsan would destroy the leukocytes or not. The patient recovered after 10 doses of this treatment, and for three months was perfectly well, with normal blood count. At the end of this time she had a remission, and upon examining her blood it was found to show 1,800 whites and 12 per cent polys. The same treatment was again instituted, but the patient died on the ninth day of illness. I feel that these patches in the throat are not caused by any organism, but by the lack of the essential elements supplied by the white cells.

DR. JARECKY: At the Sydenham Hospital last year, we had a case of this disease. The patient was a middle-aged woman, somewhat overweight, who used coffee, alcohol and tobacco ad libitum. She had an attack of Vincent's angina. The blood picture showed leukopenia. The treatment consisted principally of transfusion and salvarsan. The patient made an excellent recovery.

DR. ROSENTHAL: The Vincent's spirillum is a secondary invader on necrotic areas. In cases of so-called Vincent's angina the blood picture shows a leukocytosis. About half of the cases of agranulocytosis showed the presence of spirilli, and other cases showed streptococci and other organisms, which are probably more important. In certain cases the necrotic membrane may resemble that of diphtheria. In fact, agranulocytosis may simulate any type of sore throat. The marked depression of the leukocyte count is the important differential factor.

The patients in our series that have recovered have had very little treatment. They recovered spontaneously, excepting one case that had neosalvarsan and transfusion. Most of the cases that were fatal were treated with transfusion and salvarsan. According to literature, the neosalvarsan has sometimes been effective, and transfusions are sometimes ineffective. The disease runs a spontaneous course; and often causes death as the result of an intense widespread bacterial invasion.

Vascular Fibroma of a Teratomatous Nature, of the Nasal Cavity and Nasopharynx. Dr. J. E. MacKenty.

D. N., age 15 years; admitted April 11, 1929; discharged July 21, 1929. Diagnosis: Vascular fibroma of a teratomatous nature of the nasal cavity and nasopharynx.

History: Intermittent bleeding postnasally during past three years, at times quite profuse; increased in severity during past two months and very profuse past week. At times pain across forehead and double nasal blockage.

Examination: Septum lying against right nasal wall. Nasal spaces filled with obstructing mass, which bled profusely on touching. Mass filled entire left nasal cavity and bulged backward and downward to fill nasopharynx. In nasopharynx it appeared as pale, rounded, smooth growth; in nose as ulcerating growth.

April 13, 1929: Two 50 m.g. radium tubes applied to tumor for six hours. April 26: Two 50 m.g radium tubes applied to tumor for 18 hours. May 13: Growth smaller, but on examination of tumor mass, severe hemorrhage resulted. Ulcerating. May 22: Bleeding from both sides, but chiefly left. Foul odor. Constant frontal headache. Progressive weakness. Febrile reaction. The tumor had been bleeding profusely at intervals ever since admission to hospital, necessitating continual nasal packing and five transfusions prior to operations and five during and after operations.

Operation, May 22: Both external carotids tied with branches. No effect on bleeding. Wound in neck failed to heal on account of the septic condition of the patient. It became evident that something radical would have to be done or the patient would die.

Operation, May 28: 1,000 c.c. of blood was given just before the operation. Then, in order to control the bleeding from the operative field, we made an effort by opening the old wound on the left side of the neck to place a ligature around the common carotid, intending only to compress the artery during the period of operation. When we opened the neck wound, we found the whole area to be necrotic. The stump of the external carotid broke off, producing what would have been a terrible hemorrhage if we had not plugged the hole with the finger. We were obliged to make an incision lower down and tie off the left common carotid. The face was opened into the ethmoid region and the bone removed, so as to give a good view of the anterior nasal space. The growth filled everything in sight. It was impossible to remove it entirely in front. The soft palate was split and a little of the bone of the posterior end of the hard palate removed so as to get at the growth from behind. We found it attached all the way along the base plate and to the vault of the pharynx. Even with a tied common carotid the bleeding was considerable and controlled with difficulty. When the operation was finished, the patient was practically moribund; 1,000 c.c. more of blood was given and the patient revived immediately; 2,000 c.c. of blood were given in two hours.

The patient was in the hospital until July 21, three months in all. Had double acute ears, which healed. Continuous temperature between 100° and 106°. Though blood culture failed to show growth, I cannot but believe that it was positive. Wassermann negative. Hemoglobin from 37 to 64. Neither the face wound or the palate wound healed. Both soft tissues and bone sloughed. Not only did the palate break down, but the bone of the palate necrosed, leaving a tremendous dehiscence. The opening in the face is as you see it. No tissue left in the palate for repair. Therefore, false palate was put in. We have not closed the face wound because we are not sure whether or not the growth will recur. We are keeping it open for observation. Later, a plastic will be done on the face.

Question: Is growth returning? Is radium now indicated?

DISCUSSION.

DR. JOHN MCCOY: I saw a similar case some years ago, a boy age 10 or 11 years, who came in with a growth from the nose. On investigation, I found a growth which involved the whole pharynx, extended into the left side of the maxillary antrum and protruded from the left side of the nose. Our first procedure was to do a radical operation; make an incision anteriorly on the cheek, then open into nasal antrum and through the antrum and get a large opening into the antrum and nasopharynx. Through the opening we could see the growth and the base from the sphenoid bone. We touched the base of the growth with 50 per cent nitrate of silver solution. He was then treated in the ordinary way and we did not see him for more than two or three months, at the end of which time it was filled up again and bleeding profusely; so that

after several packings our next procedure was to use the high frequency current, which seemed the solution of the difficulty. It was used several times, going back to the base of the growth in the pharynx, and it finally stopped growing. The boy had perfect restoration.

Adamantanomatous Cyst of the Mandible. Operation. Dr. T. Morley Smith.

The patient, a deaf mute woman, age 61 years, was first seen Aug. 10, 1929, complaining of a mass in the left lower jaw, accompanied with slight pain on chewing and intermittent pain in the left ear and side of the neck.

About 2½ years previously, the patient had a swelling in the lower jaw, and two abscessed teeth were extracted. The jawbone did not heal kindly and some six weeks later the dentist curetted the cavities, following which the jaw healed, but a slight swelling persisted. This was probably the beginning of the growth. Her past history is otherwise irrelevant.

About a year later, this mass began to grow more rapidly and to cause some local soreness and some difficulty in eating. From this point the mass is said to have persistently increased in size, and about two years from the time it was first noticed she consulted her family physician, who, thinking it to be an abscess, incised it on the buccal side and obtained a small amount of dark, bloody fluid.

The mass continued to grow slowly but persistently, with coincident difficulty in mastication and quite persistent pain in the ear and tonsil region on the affected side. Her physician again incised it, with the same result as on the previous occasion.

When the patient came under observation in August, 1929, there was a fairly well defined, nonfluctuating, slightly tender, firm mass in the body of the mandible, the anterior boundary being about 4 c.m. from the symphsis. The mass was approximately the size of a hen's egg, and apparently involved the entire structure of the mandible. The pain in the ear was quite severe, and she had evident discomfort and difficulty in chewing. There was a feeling of unevenness in the edges of the mandible. There were a few palpable glands in the anterior triangle of the neck. The patient appeared to be moderately cachectic, and had lost 15 pounds in weight in three months. Examination of the nose, accessory sinuses, throat and ears revealed nothing unusual nor did a general examination. The blood Wassermann was negative, and the urine and blood were essentially normal. A Roentgenogram of the jaw showed considerable destruction, involving the upper border of the jaw in the area of the third molar, also the lower border extending through the cortex of the bone. It was reported to have the appearance of osteomyelitis or probably malignant. The consensus of opinion at this time was that we were dealing with a sarcoma, and as the patient refused operation she was referred to the radium department for treatment.

She received her first treatment at the end of August, and had four applications in all, up to the beginning of November. The radium was applied externally. In about three weeks after the inception of this treatment it was noted that the mass was growing more rapidly; it continued to do so, and by the end of October it had attained the size of a grapefruit, the mass almost filling the cavity, so that she could take only fluids with great difficulty, and the pain in the ear was persistent and severe. An exploratory puncture through the buccal side was done, and 50 c.c. of bloody, sirupy fluid obtained. The mass partially collapsed, but refilled in 24 hours. Examination of this fluid gave none of the reactions of salivary fluid.

An X-ray examination of the chest at this time failed to show any evidence in the lungs or ribs. There was a mass in the upper mediastinum and lower neck, pushing the trachea over to the right. The Roentgenologist did not venture an opinion as to what this mass might be.

Operation was consented to, and on Nov. 20, under general anesthesia, the left external carotid was ligated. An incision was then made, beginning about 3 c.m. from the symphysis and extending parallel to the jaw and beyond the angle. On going through the external wall of the mass the true cystic character was evident. The wall was about 3 m.m. in thickness and was dense and quite firm. The large cystic cavity was filled with the dark, bloody fluid previously mentioned. The cyst involved a large portion of the body of the

mandible and the entire vertical ramus up to and including the mandibular joint, which was partially destroyed. The lining was smooth and very vascular. The beginning of the tumor wall was well defined, and the bone was divided beyond the point and all that remained of the mandible, a spicule of bone 4 x 0.5 c.m., was removed, and the remainder of the cyst wall piecemeal. It was densely adherent to the underlying structures. This left a very large cavity, which was closed by interrupted sutures to a medium-sized rubber drainage tube. The neck wound healed per primam, and the jaw wound was completely healed in two weeks. There was very slight postoperative reaction, and the patient took semisolids freely from the fifth day, with practically no difficulty.

There is only a slight deformity, and her general condition at present is excellent.

The pathologist's report is as follows: "The specimen consists of a dense ovoid cyst 2 m.m. in thickness. On one pole is a section of the ramus mandibuli, measuring 1.9 x 1.2 c.m. The inner wall of the cyst is grayish-white, shows a hemorrhagic membrane and a ridge of bony tissue, the remains of the ramus. This measures 4 x 0.5 c.m. In other areas of the cyst wall there are islands of thin, membranous, bony tissue.

"Microscopically: Section reveals an inner lining of stratified squamous epithelial cells with some papillary hyperplasia, but no signs of infiltration or active mitosis. The parchment-like cyst wall is chiefly composed of dense hyaline connective tissue, with some mononuclear reaction in areas. In some of the sections thin areas of bony or osteoid tissue remains, showing osteoclasts and giant cells. At the junction of the cyst and bone there are several smaller cyst cavities lined with squamous cells. Diagnosis: Adamantanomatous cyst."

The slide (which will be shown) is typical of this type of tumor. These are comparatively rare benign tumors and, as in this case occurring in a woman over 60 years, may cause some confusion in arriving at a correct diagnosis. It is also interesting to note that, coincident with the application of radium, the tumor grew very rapidly.

DISCUSSION.

DR. A. A. EGGSTON: A few lantern slides of the microscopical sections of the cyst in this case show the detail nature of the growth. The first slide, taken from the region of the adjacent cancellous bone and cyst, reveals absorption of the bone and the presence of numerous small cystic cavities, which may be multilocular cysts or simply diverticular of the large cyst. It will be noted that the cysts are lined with stratified squamous cells and the lumena contain desquamated cells and granular debris. The character of cells present and the history of the condition following the extraction of a tooth would suggest the origin to be from the invagination of the gum epithelium. However, some of the cells are columnar and suggest the origin of the paradental enamel epithelial remains. The other slides show beautifully the process of bone absorption in the parchment-like wall of the cyst, i. e., numerous osteoclasts and multinucleated giant-celled osteoclasts with some osteoporosis.

Third Nerve Paralysis, Associated with Posterior Ethmoiditis and Sphenoiditis. Improvement Following Operation. Dr. John D. Kernan.

Personal History: The patient was born in Austria 36 years ago and came to the United States 20 years ago. Occupation, butcher.

Past History: Typhus at the age of 3 months, but has never been sick a day since except for rare slight cold until the present illness. He has had paroxysms of sneezing since childhood, but no cough or nasal symptoms. For the past few years he has been dyspneic on exercise and has had "hard breathing" for the past year or so. He has always been overweight.

Present Illness: Nine years ago, he noticed twitching of the left eyelid. Two days later, he developed diplopia, and the same day his eye closed. Then the eye began to protrude so that he could notice the increase from day to day. At the same time he had a marked and rapid swelling of the tonsils so that he almost choked. He had no sore throat, fever, headache or any other general symptoms except drowsiness. Apparently no facial muscles were involved.

He went to a doctor, who took out his tonsils; but the eye did not improve. He went to many doctors, who told him that his eye was paralyzed, but said they could do nothing for him. His sinuses were not investigated. The condition lasted for six months, during which time the only treatment was hot applications to the eye. At the end of this time all symptoms disappeared and the eye looked and moved normally.

In February, 1928, he had a recurrence with exactly the same symptoms and speed of onset, except that this time the throat was not affected. Drowsiness also returned. This time he was sent to a neurologist. Lumbar puncture, Wassermann and neurological examination were all reported negative. After this, he wandered from doctor to doctor without getting relief, and finally stopped treatment until December, 1928, when he went to Dr. J. N. Evans. One doctor had washed antra and frontals in October, 1928, and reported washings negative. This was the first time his sinuses had been examined. Dr. Evans reported partial paralysis of the left internal rectus, congestion of the retinal veins, blind spot enlarged, visual field satisfactory, and sent him to Dr. C. G. Crane. Dr. Crane found the patient's septum deviated to the left in contact with both turbinates, with a ridge formation embedded in the inferior turbinate. There was moderate discharge on the left side. The nasopharynx was inflamed. There was complete occlusion of the left middle meatus. The postoperative tonsil condition was satisfactory. The right eardrum showed loss of light reflex, and the left showed loss of reflex and chronic congestion. There was some tenderness over the left frontal and the left antrum. Transillumination showed small illumination of the right frontal and none of the left frontal or either antrum. An X-ray taken Dec. 19, 1928, showed a cloudy left frontal, left ethmoid and left antrum, both optic canals normal, equal and slightly smaller than average. On Dec. 28, he operated on the septum, left ethmoid and left antrum. Jan. 2, 1929, some improvement in the eye was noted. Following the operation, there was marked intranasal swelling and profuse discharge of pus from the left antrum, but the eye condition did not seem to make any further improvement. He was seen again in March, 1929, at which time the eye condition was the same. At this time, the left ethmoid was fairly satisfactory and some mucus was washed from the left antrum.

April 15, he went to Dr. Denig, who found approximately the same eye condition as had Dr. Evans. X-rays were taken and showed the right frontal well developed, deep and hazy. The left was smaller and cloudy. The right ethmoids were fairly clear, the left very cloudy. The right antrum was hazy and the left cloudy. Sphenoidal region cloudy. He was then sent to me.

At this time there was a little proptosis of the left eye and he complained of a little pain across the bridge and in the left forehead region. Transillumination showed the left frontal and antrum to be dark. The middle turbinates were out. Several masses resembling polyps could be seen in the posterior ethmoid region. There was pus in the nose.

On May 14, an external ethmoidectomy was done under ether at the Presbyterian Hospital.

Procedure: The usual curvilinear ethmoid incision was made from the inner angle of the eye along the side of the nose. Bleeding was profuse and was controlled by clamps. The periosteum was elevated with an elevator. The ethmoid cells were opened with a mallet and chisel. The opening was enlarged with an ethmoid punch. The lateral nasal wall was partly removed to aid exposure by means of punch forceps. Polypoid membrane was removed with a curette. The anterior sphenoid wall was opened up and thickened polypoid mucosa was removed. A counter-opening was made through the nasal mucosa. A small cigarette drain was inserted and another passed through the opening in the nose, and the wound sutured.

Pathology: The mucous membrane of all the ethmoid cells and the sphenoid sinus was diffusely thickened and polypoid in nature. No pus or tumor tissue was encountered.

Pathological Report: The specimen consists of numerous small pieces of bone, 1 m.m. thick, with a little soft tissue enclosed in the bony capsule.

Microscopic: Sections have trabeculae along the side of the section. The marrow tissue is more fibrous than usual. The opposite side of the section is

covered with stratified columnar epithelium. The intermediate areas is made up of loose connective tissue densely infiltrated with lymph. The epithelium is hypertrophied and extends into the submucous tissue in irregular projections. In the depths there are numerous well formed tubercles, some with central giant cells and some with caseation necrosis.

Diagnosis: Tuberculosis of the ethmoid. This report was rather a surprise. I have not seen many tuberculous cases in that region.

The day following operation, the patient's temperature rose to 103° and he was cyanotic and coughing. X-ray showed bronchovesicular markings throughout the lung fields. The right leaf of the diaphragm was higher than is usually seen. No areas of consolidation of infiltration in either fields to suggest pneumococci but on auscultation a patch was found in the left lower lobe. Sputume showed Type 3 pneumococci. There was a rapid drop in temperature the next day, and from then on the recovery was rapid. Sutures were removed the third day after operation, at which time some swelling of the eye was noted. The wound was clean. The patient left the hospital on the fifth day.

He was treated in my office at short intervals, and on May 27 the eye began to open, and on June 21 it was entirely open and has stayed open ever since. It is no longer prominent. Oct. 9, there was swelling and thin pus in the left sphenoid region, and some pus was seen on Oct. 26. On Nov. 26, Dr. Denig reported much less diplopia. Of all the muscles of the left eye which had been affected, only a slight paresis of the left internal rectus was left. Dr. Denig is of the opinion that this will entirely disappear in time.

At this last visit to my office, on Dec. 14, he was doing well and there was no sign of pus on washing the sphenoid. Drowsiness disappeared when his eye began to stay open.

DISCUSSION.

Dr. J. Ivimey Dowling: I wish to complment Dr. Kernan upon the brilliant achievement in this most interesting case. For years I have been interested in the relationship of accessory sinus disease to the various symptoms resulting from third nerve involvement, which may be present in simple hypertrophic or suppurative sinus disease, either without or with some complicating systemic disease, such as pulmonary tuberculosis, syphilis or diabetes. My observations and results of appropriate treatment or operation leads to the dictum that in the presence of ocular or third nerve involvement there is likely some mechanical fault that interferes with the ventilation and drainage of the accessory sinuses.

These cases are of interest to the rhinologist and oculist because, despite any systemic complications, symptoms arising because of third nerve, and sometimes sixth nerve, lesion may be overcome through conservative or radical surgical methods directed to the end of providing ventilation and drainage of the accessory sinuses.

I have been successful in accomplishing cosmetic cure of the extreme types of divergent and convergent alternating strabismus through intranasal surgery such as the doctor has described as successful in his case.

The important point I desire to make is: In the presence of third nerve involvement, complete systemic treatment by appropriate measures directed to the relief of the possible sinus disease.

Furthermore, the internist, rhinologist and oculist should work together more harmoniously, for they one and all are simple complements one to the other.

In arising to discuss Dr. Kernan's case I merely had the thought of emphasizing the importance of correct surgery in types of cases such as he has described. The Society as a whole is indebted to him for this accurate and valuable report.

Abscess of Frontal Lobe, Simulating Frontal Sinusitis; Presentation Nine Years After Operation. Dr. Francis W. White.

October, 1919, girl age 11 years came to the Manhattan Eye, Ear and Throat Hospital complaining of headache and a constant desire to sleep. Left upper eyelid swollen.

Previous History: Seven years previously, a compound fracture of the skull. Since then has had a great tendency to nausea and vomiting when riding in

cars. Excessive light caused severe headache. Mentality low. Ungraded class of public school. Lost her ability to read.

Present History: Two weeks ago, severe pain in the head and convulsive attacks. High temperature. Somnolent. Keeps her eyes closed all the time. Three days before admission, swelling of the left upper eyelid.

Examination: Patient is lethargic and resents being disturbed. Eyes closed. Left upper eyelid edematous and reddish. No ocular palsy. There is a scar about 6 inches long, extending from the middle of the forehead, slightly to the left of the median line. Marked tenderness on pressure over the frontal sinus. No meningeal symptoms. X-ray shows marked cloudiness of the left frontal sinus, which is small. Eye examination negative. Temperature 98°.

After three days of conservative treatment there was a sudden rise in the temperature to 101° and immediate operation was decided upon. Under ether, the usual procedure was carried out in opening the frontal sinus. Free pus and granulation tissue were found. The entire posterior wall of the sinus had been eroded. The dura was dark and very friable. Probe introduced without pressure went backward, paralleling the cribriform plate for more than 1 inch. A vertical incision in the tissues of the forehead was made joining the primary incision, thus forming a triangular flap. This was dissected off and the anterior wall of the sinus removed. While removing the rough edges of the bone, greenish-yellow pus escaped from the frontal lobe; probably about an ounce. The abscess cavity did not collapse, due to the thickness and rigidity of its walls. The cavity was 1 x 0.75 inches. Drainage was instituted. Cultures from the pus showed no growth after 120 hours. The day after the operation the lethargy disappeared, but thereafter the patient exhibited a very violent temper. Within four weeks the patient was discharged, apparently cured.

Three months later, the principal of her school stated in a letter that she had been irregular in attendance, that her work showed no improvement, but that her general demeanor was much better.

Comments: This case seems to be of peculiar interest, in that the fibrous capsule of the abscess was more resistant to pressure than the skull. There seems to be no question that the abscess was due to the injury seven years previously and had been dormant for years. It is difficult to say just what caused the increase of its contents and consequent pressure.

The above is a resume of Dr. Samuel McCullagh's paper entitled, "Abscess of the Frontal Lobe Simulating Sinusitis", as it appeared in the Transactions of the American Laryngological, Rhinological and Otological Society, Inc., 1920.

One year later—October, 1920—the patient was readmitted, complaining particularly of headache. She was cross and irritable. Somewhat somnolent when not disturbed. Photophobia. There was marked rigidity of the neck. Kernig sign marked in both legs. Tache cerebral present. By the next afternoon all signs and symptoms had increased markedly. White cells, 28,000. Spinal fluid very turbid. Sterile. The patient was operated upon that evening. An incision was made in the line of the scar from the previous operation. The fibrous tissue separated and the periosteum was elevated away from the edges of the bone. Projecting edges of bone were removed and the area corresponding to the form of the frontal sinus cleaned out. A very small quantity of creamy-white pus was discovered and a culture made. Fibrous tissue reaction was marked. Exploratory punctures were made into the brain substance in all directions, but no more pus was discovered. Drainage was instituted and a wet dressing applied. During the night, the temperature reached 105.2° but the next day receded to 100.2°. Immediately following the operation, the patient was much brighter, responded quickly to questions and did not complain of headache. By the fifth day, rigidity of the neck had disappeared and the Kernig sign was practically nil. For three days after the operation the temperature reached 102° or 104°, to fall to normal within 24 hours. Then, 10 days later, the temperature, not having gone over 100°, suddenly reached 105°, and the next day dropped to 100.4°. Three days later, the temperature was 104° and then rapidly fell to 99.2°. This was repeated until nine more almost similar rises and falls of temperature occurred on consecutive third and fourth days. The temperature did not rise again above 100° up to the time of her

discharge, Dec. 1. With the sudden rises of temperature the patient complained of very severe pains in the lumbar region and upon one or two occasions it became necessary to administer an opiate. At no time had the patient chills or sweats. Repeated examinations of the blood for plasmodium malariae were negative; however, quinin supplemented by Basham mixture and Fowler solution was administered.

Laboratory Reports: Oct. 13, day of operation: White cells, 28,000 (highest). Spinal fluid, 14 c.c. Very turbid, albumin heavy trace, globulin heavy trace, sugar present, acetone negative, lactic acid heavy trace, bacteria none. Oct. 14: Blood culture negative, after 18 hours. Culture from brain abscess profuse growth of streptococcus capsulatous.. Oct. 23: White cells, 12,000; malaria negative. Oct. 29: Blood culture negative, 20 hours. White cells, 19,400. Malaria negative. Nov. 4: White cells, 12,400. Nov. 16: White cells, 6,400. Nov. 22: X-ray. No spinal involvement; moderate involvement of right antrum, other sinuses clear.

Carcinoma of Neck Extending Through Thyroid Cartilage; Second Showing of Case. Dr. Arthur S. Wilson.

A. J. P., age 40 years, was first admitted to the Manhattan Eye, Ear and Throat Hospital, clinic of Dr. MacKenty, in August, 1925, with a negative family history, but a personal history of frequent sore throat, hoarseness, colds and attacks of quinsy since the age of 20 years. In March, 1922, following an attack of influenza (?) and quinsy sore throat, he became extremely hoarse and his voice was reduced to a whisper for several weeks at a time. In July, 1925, he noticed a slight transient difficulty in breathing.

On examination, pus was expressed from the tonsils; the mucous membrane of the larynx, including the vocal cords, was rusty-red and congested; the cords were slightly thickened. A sputum examination was negative for T. B. X-ray examination of the chest and Wassermann test were negative.

A tonsillectomy was performed under general anesthesia on Sept. 24. The larynx became edematous a few days after the operation, and a tracheotomy was performed on Sept. 24. Following the tracheotomy, the edema of the larynx gradually subsided, leaving the larynx in about the same condition noticed at the first examination; but the vocal cords were motionless and the tracheotomy tubes had to be retained.

In 1926, during a very violent coughing spell, the patient experienced a sharp pain in the neck to the right and just a little below the tracheotomy tube; following this, there was redness, pain and swelling of the neck around the tracheotomy tube, mostly on the right side. The family physician sent him to the Manhattan Hospital, where the abscess was opened by the house surgeon. A fistula formed and the house surgeon brought him to me for advice. Examination of the larynx at this time showed edema of all parts of the larynx, and this gradually increased until the larynx was completely blocked. The fistula was opened three times during 1927, at about three-month intervals. At the last operation, one of the fistulous tracts was traced to the right side of the thyroid cartilage, which was necrotic. At this time there were several fistulous tracts between the tracheotomy wound and the thyroid cartilage, all discharging pus. A diagnosis of chondritis of thyroid cartilage following tonsillectomy was made.

During 1928, the patient was hospitalized twice and pockets in the neck were opened. Examination of the chest was negative for T. B. He developed a condition of chronic toxemia and a laryngectomy was performed on June 28, 1929. At the operation, the right side of the thyroid cartilage was found to be completely necrotic and there was a red ulcerating growth on the right side of the trachea, extending from just below the thyroid cartilage inferiorly to the substernal region. This growth involved the trachea, esophagus and the adjacent tissue, and was removed as completely as possible.

Pathological examination proved this to be a squamous-cell carcinoma.

A large area of the esophagus was removed and the patient has a permanent esophageal fistula in the neck. He has been treated with X-rays at the St. Luke's Hospital, but with no benefit as the growth is slowly increasing in size.

Probable "Sequence of Events": A chondritis following tonsillectomy, due

to an acute exacerbation of a chronic inflammatory condition of the larynx; a necrosis of the right thyroid cartilage; a slow seepage of necrotic material along fascial planes, with a blind fistula; a rupture of this blind fistula during the severe coughing spell in November, 1926; infection of the adjacent tissue, with pocket formation and the development of a slow-growing carcinoma as a result of the irritation of the pus.

Plasmocytoma of the Nose. Dr. Harry Rosenwasser.

Published in full in THE LARYNGOSCOPE, *August, 1930.*

Transitional-Celled Carcinoma of Larynx, Three Years After Radium Treatment. Dr. G. Allen Robinson.

M., age 55 years, had complained of hoarseness and difficulty in swallowing coarse foods, which began three months previous to admission to Manhattan Eye, Ear and Throat Hospital in May, 1927. He had lost 15 pounds in weight and the swallowing was becoming more difficult.

Examination showed a large vascular tumor mass involving the posterior part of larynx and arytenoid areas. Biopsy revealed reticulum-celled sarcoma.

Radium treatments amounting to 19,850 m.g. hours were applied to the larynx on either side, in divided treatments, over a period of several months. The growth gradually disappeared, patient regained his normal weight, and the voice became normal.

In November, 1929, patient noticed a small tumor mass on left shin, which gave him no pain nor interfered with his walking. Four weeks later, a swelling on the right forearm was noticed. The tumor mass over fibula measured 7 x 4 x 4 c.m., firm, elastic nature and no limitation in ankle joint was noticed. The overlying skin was normal. The circumference of the right forearm was 5 c.m. greater than the left. The larynx appeared normal except for slight redundance of mucous membrane in the region of the right corniculate cartilage. Aspiration of right forearm for biopsy revealed a transitional-celled carcinoma, which resembled closely the primary lesion in the larynx. There is a difference of opinion among pathologists whether or not this type of tumor is of lymphoid or epithelial origin.

Deep X-ray therapy, at the Memorial Hospital, applied to the metastatic areas resulted in a prompt response after seven to 10 days consistent with the response by radium therapy on the laryngeal growth. This case is of unusual interest; first, because of the highly malignant and unusual neoplasm of the larynx; second, the good result by radiation and a period of good health for nearly three years; and third, unusual metastasis to forearm and ankle.

DISCUSSION.

DR. DAVID H. JONES: When the patient came to the hospital he had edema of the epiglottis and arytenoids and presented a clinical picture of tuberculosis. X-ray examinations of the chest, sputum and Wassermann were negative. A number of biopsies were done and the last report was round-cell carcinoma of the lymphoid type. Dr. Robinson began his treatment with radium blocks and it was remarkable to see the results.

At the present time the examination of the larynx shows no involvement of either epiglottis or arytenoids, and his cords show no sign of paralysis, voice being normal.

Pemphigus with Initial Lesion in the Pharynx. Dr. Louis Hubert.

E. G., a girl, age 26 years, Hebrew, born in United States, came to see me on Dec. 25, 1928, complaining of a severe pain in the throat. Examination of the throat revealed two small, grayish-white areas on the posterior wall of the pharynx, which at first glance appeared like two mucus patches seen in syphilis. Looking more closely, I noticed that the patches were eroded and slightly macerated, with some shreds around their margin. I thought that they were ruptured herpetic vesicles. After the application of a 10 per cent solution of silver nitrate to the patches, the pain was very much diminished. On Jan. 13, 1929, the pain returned with greater severity. At that time there were several ruptured vesicles on the anterior pillars of the fauces and the posterior pharyngeal wall. Again the pain was relieved with silver nitrate. On Feb. 19, the patient had a severe chill, followed by a temperature of 105°

and excruciating pain in the throat. She was examined by her family physician, who saw white, diphtheroid patches over the tonsils and pillars, and therefore thought of diphtheria. The culture taken from the tonsils showed numerous streptococci. The high fever continued for a week, then dropped to 99°, and ranged between 98° and 101° throughout her illness. After the acute attack, which we thought was a streptococcic sore throat, was over there appeared numerous macerated patches on the oral mucous membrane, the tongue, gums and lips. Some of these patches were white, surrounded by a red areola, others were merely erosions with epithelial shreds about their margins, thus showing their bullous character. Actual bullae on the mucous surfaces were never observed. The pain the girl was suffering was atrocious. The only thing that seemed to relieve it was the application of a 4 per cent solution of chromic acid to the eroded areas. About the middle of March, the patient developed an eruption on the scalp, which emitted an extremely offensive odor. A dermatologist thought that this was a streptococcic infection. At that time the oral cavity cleared up somewhat so that she could take some nourishment. Four weeks later, when the scalp eruption was practically gone, the oral cavity became worse again. She was seen by an oral surgeon, who was certain that the girl was suffering from Vincent's angina. The usual treatment for that condition was of no avail. About the middle of May, the girl developed a bullous eruption on her body, and practically all mucous surfaces, including the rectum, vagina, the nose and conjunctiva, were involved. I was then considering the possibility of pemphigus and referred the girl to a prominent dermatologist, who had considerable experience in that disease. He thought that the girl was suffering from an erythema multiforme bullosa, and advised intravenous injections of sodium salicylate. On May 20, the patient was admitted to the Manhattan Eye, Eye and Throat Hospital. There she was examined by Dr. Howard Fox, whose report was as follows: "From the patient's race (Jewish) the history of the eruption first affecting the mouth, the loss of weight (22 pounds), and a nonitching vesiculobullous eruption of the cutaneous surface, a diagnosis of pemphigus vulgaris is strongly suggested." Dr. Fox also suggested that a specimen of the patient's blood be sent to Dr. Pels, of John Hopkins, who is working on a test for pemphigus, which looks promising. The test is known as the phytopharmacologic test and depends on the action of serum on a growing seedling, Lupinus albus. Pels and Macht have shown that the serum from patients with pemphigus are definitely toxic for the growth of these seedlings, and that the serum obtained from patients with other skin diseases and from normal individuals are not toxic. Macht has also shown that in the blood of menstruating women there is a toxin which is deleterious to the growth of these seedlings. Therefore, the blood for testing of pemphigus should not be taken during the menstrual period. The report received from Dr. Pels was positive for pemphigus. Other laboratory tests done by Dr. Eggston were as follows: "Stained films and culture from mouth show pus and epithelial cells and mixed bacteria, chiefly streptococci. There are a few spirochetes and fusiform bacilli. The slide is not typically one of Vincent's angina. Culture shows a very profuse growth of streptococcus viridans. Wassermann and Kahn reaction negative. Blood count showed 3,339,000 red cells, 12,400 white cells. Hemoglobin, 71.5 per cent. Polymorphonuclear neutrophiles, 82 per cent; small lymphocytes, 17 per cent; large mononuclear, 1 per cent; immature polynuclears, 12.5 per cent.

She stayed at the hospital only a few days and was taken home and slowly wasted away, a horrible picture to look at. Her mind was clear practically until the end, when she died on Dec. 7, 1929.

Comment: We must remember that pemphigus often begins in the pharynx, mouth, lips and nose, and that the manifestations in these localities may precede the cutaneous lesions for weeks, months and, exceptionally, for years. In this particular case, five months elapsed before a vesicular eruption appeared on the cutaneous surfaces. We must also keep in mind that bullae are very seldom seen on the mucous membranes, as they rupture almost immediately and produce eroded and macerated patches, which are difficult to distinguish from syphilis, ulceromembranous stomatitis, Vincent's angina and diphtheria. If the patches are carefully observed we will find the persistence of epithelial shreds

about their margins, which shows that they were originally bullous and can thus be distinguished from the above-mentioned diseases. However, exactly the same lesions may be found in erythema multiforme, drug eruptions, dermatitis herpetiformis, herpes zoster and herpes simplex. The differentiation between these diseases is not only a question of academic interest, but often involves the life of the patient. Pemphigus is always fatal. The other diseases mentioned are amenable to treatment. It used to be said that if the patient dies it is pemphigus; if he recovers it is some other skin disease. The phytopharmacologic reaction of Pels, if proven to be reliable, will therefore be of the utmost importance. In this case the diagnosis of pemphigus was made in Baltimore by testing the blood of the patient; while the New York clinicians were doubtful up to two or three months before her death whether or not they were dealing with a case of pemphigus, erythema multiforme bullosa or dermatitis herpetiformis. Before the bullae appeared on the body, most of the consultants thought that the girl was suffering from Vincent's angina. Pels has, therefore, opened up a new line of investigation in a disease which has hitherto been clothed with obscurity so far as all methods of investigations have shown.

DISCUSSION.

DR. L. M. HURD: Pemphigus, as we all know, is a very rare condition. I remember a case that was suspected for a year, and then one day we found a large vesicle in the pharynx; after that we lost sight of him. A couple of years ago I saw a patient who had a large lesion in the mouth for a year, left eye was involved with terrific pain in the eye, which was enucleated. The pharynx and inside of the cheek had become very much indurated. The only thing that gave relief was a mild application of radium. Another case, that of a man, age 55 to 60 years, sent to me complaining of a soreness of the larynx when he swallowed. I found a white patch such as Dr. Hubert has described, and did not know what it was. He informed me that he had lymphatic leukemia. Three or four days later, he sent for me again with considerably more sore throat, and I found a number of blebs in the pharynx, larynx and soft palate. I don't know anything about the laboratory tests, but you do find these enormous blebs in the mucous membrane. The man wanted to take an automobile trip, and I agreed. When he came back they sent for me again, and the eruption covered the nose and throat mucous membranes, and all over the skin; it looked like chickenpox. Such a diagnosis was made by the local doctor, but it was acute pemphigus and the patient died within a week.

DR. LOUIS HUBERT (closing): I only wish to say a word about Vincent's angina. This girl had been seen by a number of men, and because they found some Vincent's organisms around the roots of the teeth they thought it was a case of Vincent's angina. Dr. Eggston has said that you can get Vincent's organisms around almost anyone's teeth, and before making a diagnosis of Vincent's angina you should get a typical picture of the spirilli and fusiform bacilli from the ulcerations of the oral mucous membrane.

SECTION OF OTOLOGY AND LARYNGOLOGY.

Stated Meeting, Feb. 6, 1930.

Observations and Results in Adenoid and Tonsil Surgery. Dr. David H. Jones.

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DISCUSSION.

DR. WILLIAM P. ST. LAWRENCE: In discussing the indications for removal of the tonsils, it is wise perhaps to divide all cases into two groups: those which present local indications, and those which present indications concerned with systemic disease.

Taking up first the question of size: All large tonsils are not necessarily diseased and the tonsils present an actual obstruction in only a small percentage

of cases. At birth the tonsils are small and remain so, as a rule, throughout the first year. Between a year and 18 months, in this climate, they tend to enlarge and this enlargement is appreciable, lasting often through the fourth or fifth year. At this later period many cases tend to recede and are actually small at older ages. Obviously, the removal of tonsils at this period, from the standpoint of size alone, would be unnecessary. On the other hand, it is well known that small tonsils are often seriously diseased and their removal may be accomplished with exceedingly satisfactory results.

Concerning the question of infection: This may be made definite by the recurrence of small amounts of exudate in the tonsillar crypts or by an almost constant, fine, lacy network of exudate over the surface. Very often this condition is accompanied by redness of the pillars and the posterior wall of the pharynx. On the other hand, infection may be made known only by the frequent recurrence of acute inflammation of the tonsils and in the intervals between attacks the tonsils may appear small and negative to examination. So it happens that one may very often find it necessary to determine the presence of infection by the history alone.

The tonsillar nodes often tell more about the tonsils than the tonsils themselves; for, without doubt, a continued enlargement of these nodes bespeaks tonsillar infection, regardless of the appearance of the throat. Ten years ago, I presented a paper on the indications for tonsillectomy and in that article I described a number of cases in which the tonsillar nodes remained constantly enlarged before operation, yet in which these nodes diminished enormously in four or five days after operation, while the throat was still covered with a slough.

Many believe that prolonged or repeated attacks of Vincent's angina may best be prevented by tonsil enucleation. Inasmuch as repeated attacks of otitis media result from an infection in the nasopharynx, this condition would serve as an indication for the removal of tonsils and adenoids. It is also believed that recurrent sinus infection may be similarly improved.

Taking up the question of systemic conditions which are likely to be improved by the removal of the tonsils when diseased, one finds it would require a very long list, indeed. It is perhaps best to limit discussion in this respect to three sets of conditions. First, the problem of recurrent respiratory infections. The work of Kaiser at Rochester seems to be particularly valuable. He observed a large series of children between the sixth and tenth year over a period of three years. Of 1200 children there were 545 who presented indications for tonsillectomy in which colds were frequent before operation. In only 146 children were colds observed after operation during a period of three years. Of 1200 children presenting indications for tonsillectomy, but for some reason not operated upon, there were 552 who suffered from colds during the same period. It was his impression that in the age group between two and five the operation was of less value from this standpoint.

The second systemic condition concerned with chronic tonsillar infection is the undernourished state. There are many factors concerned with this problem, although, without question, the tonsils are often important. If the undernourished state is accompanied by local conditions which would speak for removal, the problem is simple. However, very often no abnormal factor can be made out. Yet, it has been observed that removal of some of these tonsils has been followed by definite improvement. It is indeed a gamble and one cannot be sure that improvement will occur, but if other measures fail, it is well to err on the side of removal.

The third condition comprises the rheumatic infections. Different observers hold different views. Personally, I believe that the removal of the tonsils early is an important factor in diminishing the recurrence. Even though recurrences are not completely prevented, much good can often be accomplished. Inasmuch as intercurrent febrile disease of any kind tends to act as an activating agent, causing latent rheumatic infection to flare into activity, it follows that any procedure which tends to remove recurrences of febrile states cannot but be followed by an improvement in the clinical course of rheumatic disease. Obviously, in a disease as widely disseminated over the body as is the case in rheumatic infection, the removal of one single focus could not be expected to

be followed by the complete absence of new attacks. I am convinced that new attacks of active heart infection and heart failure are diminished by eradicating diseased conditions in the nasopharynx.

Management of Otitis Media in Children. Dr. Edmund Prince Fowler.

I hesitated to accept the invitation to present this paper before the Sections of Otology and Pediatrics on "The Management of Otitis Media in Children", because it would seem a waste of time to go over the familiar ground fully covered in the textbooks, but your Chairman was gracious enough to permit me to treat the subject from a somewhat different angle and, therefore, I feel that maybe my effort is warranted. Condensing to a few words the general management of otitis media, I shall then discuss more in detail something about its etiology. After all, removing the cause of disease is the best treatment. The treatment and management of otitis media should have four main objectives: 1. Elevation and cure of the infection. 2. Prevention of complications. 3. Restoration and preservation of the hearing. 4. Prophylaxis for the opposite ear and for recurrence of the inflammation.

These objectives are all approached by adequate drainage through the Eustachian tube and spontaneous rupture of the drum or its incision. If repeated ruptures or incisions are necessary to obtain drainage, it is probable that there is a pus pocket not draining into the tympanic opening with sufficient constancy to maintain the flow of pus, and if the child looks sick, if general or local clinical signs indicate increasing sepsis or complications, the conservative procedure is simple mastoidectomy. Nonsuppurative and suppurative otitis media often occur in children without pain or manifest distress or discharge. I frequently see cases with adhesions and ankylosis in the tympanum in which no history of pain or discharge or other symptoms have ever been noted. It is quite customary in such cases, and in others where things have gone wrong, to say that an ear was not opened early enough. I doubt whether a few hours more or less would make much difference so far as complications are concerned. I know that proper treatment can save the hearing of those patients. The most perfectly treated ear infections may go on to mastoiditis, sinus thrombosis and meningitis, and the most negligent speedily recover with perfect hearing, so we must not condemn our confreres if their treatment does not wholly coincide with our ideas. There is one thing that should be condemned, however, and that is the rushing of patients into operations by exaggerating the immediate danger of middle ear disease and of mastoiditis. There has been entirely too much operating by otolaryngologists and a reaction is setting in. Nature will do pretty well for most cases of otitis media if we aid her by treating the cause. Excluding trauma, abscess in the ear is regularly born within the nasal pharyngeal spaces. The ear is really but one of the sinuses of the nose, and I so treat it. Failure to conquer the disease in the nasal pharyngeal spaces is the outstanding crime in otologic practice today. Even in our best clinics, how much attention is given to the nose by the ear department? How much attention to the ear is given in the nose and throat department? Very little, unless the patient demands it. This is the main reason why we have over 3,000,000 deafened children in our country today.

Treatment of the nose and its sinuses is of the utmost importance in the obtaining of the four main objectives outlined. Everyone knows that nasal inflammation may extend to the middle ear, but the extent to which the nasal sinuses are involved has not been appreciated. Eighty-six per cent of my cases, transitory, chronic or with progressive deafness, are intimately associated with disease in the sinuses, as demonstrated by Roentgenogram. I will demonstrate to you lantern slides showing the coincident variations in the hearing and in the Roentgen and clinical pathology in these cases. I will show you chronic otitis media with marked deafness recovering to average normal hearing through nonoperative treatment of the nasal sinuses. In children, nonoperative treatment has been very efficient.

In the year ending June 30, 1929, by well recognized methods, supplemented by home treatment, the hearing of over half of all the children sent by the League for the Hard of Hearing to my clinic at the Manhattan Eye, Ear and Throat Hospital were benefitted, and 25 per cent markedly so. These children

are picked up through 4A Audiometer tests in the schools. I have slides of the history charts of all of these cases, but to conserve time will show only a few as a cross-section of the data at this time. From experimental work there is considerable evidence that deficiency in Vitamins A and D and calcium phosphorus imbalance are associated with middle ear and sinus infections. These factors have, therefore, been given careful study in all the cases. Please note that every patient receives, in addition to a careful otologic examination, audiometer tests, stereoscopic Roentgenograms, blood chemistry, Wassermann and intracutaneous T. B. tests, also careful inquiry into nutritional disorders and the diseases of childhood. By tabulating all this data, it was hoped that something of advantage would be learned. I can only say at this time that much has been learned, and something of advantage.

About 15 slides of various hearing and coincident novel sinus conditions and variations were demonstrated, followed by pictures of the X-ray plates demonstrated by Dr. F. M. Law.

DISCUSSION.

DR. L. E. LAFETRA: As an impromptu substitute in this discussion for my good friend, Dr. Wilcox, I feel very incompetent after having heard the excellent paper of Dr. Fowler, covering in detail the management of deafness. I am unable to discuss that part of his paper, and so will take up only that part of his remarks with regard to acute infections, particularly from the standpoint of the pediatrist, who sees so many cases of otitis at their outset.

Undoubtedly, as Dr. Fowler says, there are many different and satisfactory ways of treating acute otitis media in children; he emphasized what I believe in heartily, that the important consideration is the matter of efficient drainage. That does not mean to the mind of the pediatrist, or the general practitioner, who sees these infants in the early stages of the inflammation, that incision is immediately necessary when the eardrum is seen to be red, or even bulging.

In the diagnosis and treatment of acute otitis media in infants and children, there are two or three things which I think are of extreme importance. In the first place, when the baby is ill with otitis media, the baby does not give you any indication in the majority of cases, that he has an earache. Not at all; he is simply sick. He may have fever, or vomiting, or apparently a pain in the stomach; it is the part of the physician, whether he be a general practitioner or pediatrist, to find out where the trouble is. That is one reason why I have always insisted, in speaking to my students of equipment for private practice on the need of a good electric otoscope. This is much more important than a stethoscope. With eyes, ears and fingers, he can much more easily make accurate diagnosis of a chest condition without a stethoscope, than he can of a condition in the ear without an otoscope. So the important thing is first to be able to see the eardrum and know exactly its condition. I have seen many young men, and some not so young, peering through a hole in a head mirror with reflected light, into a small ear canal filled with hairs, in the effort to see the infant's drum. They do not see much. Of course, the wise thing to do is to say: "The drum is red; we will open it." While with a good electric otoscope, it may be seen that the drum is not bulging at all, and with conservative treatment the whole condition will probably subside. The first consideration then is to know what the drum looks like.

I shall not consider the indications for, and the technique of, opening the drum. These are perfectly understood. I do not believe, however, that the incision of the drum is an entirely harmless procedure. I think many secondary or multiple infections of the middle ear occur due to improper or unnecessary incision of the drum. It is nearly impossible to prevent micro-organisms entering from the canal, no matter what careful technique is used. It is often wise not to open the drum without 24 or 48 hours of careful observation. As Dr. Fowler said, the condition of the nares should be watched very carefully, and a toilet of the nares done. In this way sufficient drainage may be obtained through the Eustachian tube. Some mild astringent or vasoconstrictor drug should be used, such as 1 per cent ephedrin or adrenalin in oil; if the child apparently has pain, a small amount of 5 per cent carbolized glycerin in the ear will relieve it. I know some good men object to this, but my experience

with it has been very satisfactory. If the drum membrane is watched carefully, and the bulging persists, an incision should be made.

After incision it is advantageous to wait a few moments, and then wash out the clots that form at the site of the incision, because otherwise one may not get the free drainage which should be obtained as soon as possible. Afterwards, the ear canal should be kept clean. Some prefer to use dry dressings with wicks of gauze. That necessitates frequent visits by the physician or a competent nurse, as it is usually not possible to carry out that particular treatment. If carbolized vasoline is spread on the skin of the ear canal, drainage is promoted. When the discharge tends to become thick, or to cake in the canal, then irrigations are of value. The double current glass ear tip should be used.

One must not expect the temperature to fall within a few hours after the drum is incised and the pressure relieved. It is more likely that the temperature will not fall perceptibly for 48 hours; often not until the discharge is purulent. The complication we always fear in otitis media is the development of mastoiditis. I presume that in a very large proportion of cases of otitis media, the mastoid is more or less involved by congestion or infiltration. The important thing I would insist on is this: that neither on account of temperature, nor on account of tip tenderness of the mastoid, should one make a diagnosis of operative mastoiditis. One should have definite evidences in the drum or marked tenderness over the mastoid itself.

DR. F. M. LAW: I wish to say just a few words in explanation possibly of the X-ray examinations in these cases that Dr. Fowler has presented. As you perhaps noticed on the reports made on the special charts we devised, consideration has been given not only to the degree of blocking or opacity in the sinuses, but to the character, the condition or appearance of the bony structures, particularly the ethmoid cells. Now, in order to make these interpretations, it is absolutely essential that the films or plates be made with perfect technique, stereoscopic, and of a quality which will show detail in the ethmoids similar to what you would get in a good mastoid plate. We must be able to distinguish each separate ethmoid cell.

In the course of an inflammatory process the ethmoid structure will vary in its appearance on the well made film; will vary from the thin, definite line of the normal structure to the fuzzy, filmy outlines of the diseased structure. This fuzziness is due to a swelling of the mucoperiosteum. This swelling consists of, or is indicated by, the outline of the lime salts in the bone, which are spread out over a larger area, and the distinctness of outline diminishes as the condition progresses, until the cells entirely disappear. If the condition improves, you will gradually see those cells come back into vision, and again have the appearance of the normal structure. This condition is indicated in these charts by the letter "E", in the worst phase as "EEEE", and in the intermediate conditions as "EE" and "EEE". The lime salts having been absorbed, the cells become invisible.

I should like to show a few lantern slides of these films to demonstrate the clean-cut lines of the normal bony structure in the ethmoid cells, and the filmy outlines of the progressive stages of inflammatory reaction . These three cases I am going to show are the last three Dr. Fowler reported on, in which there was improvement.

By the way, these X-ray examinations were made independent of any knowledge of the progress of the case. The case is sent in and a report on the films returned to Dr. Fowler, who tabulates it. After a certain number, he was able to make a report on his tabulations. The Roentgenologist does not know the nature of the case.

The first film shows a difference in the antra. In the next slide, after a few months, we see an improvement in the antra. From looking at the A.-P. plate, using the old type of reporting, you would say the ethmoids are normal, but they are not. In a later view, we see the ethmoid structure, showing a few cells more distinctly visible. Coincident with the improvement in these plates, the audiometer showing indicated a similar improvement in the hearing. In the next series, we see a blurring and fuzziness of outline of the cells, due to exudate and edema, while in the second plate, the outlines are distinct. As the condition improves, the outlines of the cell walls come out very much more

distinctly. In the third case, one antrum is clouded, and a definite ethmoiditis is present; the slide shows no ethmoid structure visible; it is all absorbed. On the second examination, months later, the bony structure is visible very clearly.

In every case the audiometer test varied in direct proportion to the change in the ethmoid structure.

DR. WENDELL PHILLIPS: I shall make no attempt to enter into full discussion of all the points covered by Dr. Fowler. In the first place, it seems to me that we should clearly understand that there are two distinct types of middle ear affections; one is the purulent type, and the other the nonpurulent type. It is therefore necessary to be able to tell in each individual case whether it is purulent or nonpurulent.

In my experience, the purulent types always show a reddened and bulging drum, while the nonpurulent or, as we please to call it in the literature, the catarrhal type (a misnomer in a sense) may have an extremely red drum but it never bulges. The purulent type should be incised as soon as seen, and the other, never. The treatment for the latter type should entirely refer to the nose condition, as the cause is Eustachian tube obstruction.

As far as the treatment of the acute cases, Dr. Fowler has completely covered the field. With the auditory canal as sterile as you can make it, and with one incision in the drum, you have done all that need be done, except to keep the canal as clean as possible and the drainage free. Make no more incisions, for what you do not get from the drainage in one incision, in final results, you will not get at all. Always maintain the treatment of the nose and throat.

Regarding the other phase of this topic which Dr. Fowler has brought to your attention, I do not think that Dr. Fowler made clear to you that these little patients he is treating—and they are all children—are not patients who voluntarily seek treatment for the ear condition from which they suffer. They are patients discovered during examination in the school examinations, and the social workers see to it that they fall into Dr. Fowler's hands, and he in turn is applying research methods as to how to prevent these little children from developing the progressive deafness of adults, a condition so well represented in this room tonight, and in any other room of adults over 35 years of age.

It may be of interest to you to know that the development of the audiometer and the possibilities which have come from the audiometer examinations, and the improvements in this instrument, have made it possible for us to make systematic examinations of the hearing conditions of young children throughout the country. You can imagine how difficult it would be to go into a school of a thousand children with five or six otologists to examine the hearing of these children. So Dr. Fowler and Mr. Fletcher developed the phonograph audiometer, which is an arrangement which permits the examination of children *en masse*, 40 at a time. This work, begun by the New York League for the Hard of Hearing, has spread over the country as a result of the propaganda efforts of the American Federation of Organizations for the Hard of Hearing, so that several million children have been examined in this country, and the results of this examination have been astounding. We are only able to examine children by this method above the second grade, and we have results that show defects in hearing in over three million children of the public schools of this country; and this is a low estimate. These three million children are the prospective deafened people of the future. The efforts of Dr. Fowler and his thorough work and research, I think we all appreciate. He has already brought out some very definite results, and with other work he is doing along these lines, we expect real results. I know how difficult it is to understand these charts he has shown you, but I can assure you he is doing a very basic and fundamental work which is destined to add to the comfort and happiness of future mankind.

Sinusitis in Children. Dr. Lewis A. Coffin.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. C. H. SMITH: It is unfortunate that there has not been more opportunity for discussion between the pediatricians and the nose and throat men.

Of course, in former years we were all taught that there were no sinuses in children. Looking over half-a-dozen volumes of pediatrics in my library, I

was able to find no mention of sinusitis in any book on pediatrics until 1928. I should like to read verbatim from Pfaundler and Schlossam, 1912, a few words on this subject: "A diseased condition of the accessory cavities, which plays such an important role in the chronic rhinitis of adults, need not be at all considered in young children because these cavities are very small and do not begin to participate until towards puberty."

I remember the first case I saw 25 years ago. A child of 2½ years, with fever, crying with pain in the malar region. Physical examination was negative except for some nasal discharge. The child finally developed a swelling over the antrum. It was thought impossible for the child to have sinusitis, because he was too young, but he did have sinusitis, and it was relieved by aspiration by a competent nose and throat man.

In the children's service at Bellevue Hospital, we have a class of children for observation, referred to the class because of suspicious chest signs or symptoms, or positive tuberculin reaction. Several years ago, Dr. Edith Lincoln, who was conducting this class, found a large number of children with chronic cough, who gave no positive tuberculin reaction, and who had no other evidence on examination of tuberculosis. She has separated off finally from that chest clinic, a sinus clinic, and in the last two years has collected three hundred cases of children suffering from acute sinusitis. This is in a medical pediatric clinic. The cough cases were the most prominent, and the most easily recognized. Many children with chronic cough, with few or no lung signs, in whom the X-ray shows some increase in lung markings (an early peribronchial fibrosis), on X-ray of the sinuses showed a tremendous percentage of involvement of the maxillary and ethmoid. So we have gotten in the routine of taking X-rays of the sinuses on practically every child with respiratory disease, and as a result have collected this large number of cases in a short time. In the next week after Dr. Lincoln first presented to the staff conference her results, there were half-a-dozen of us who discovered cases, following her suggestion to X-ray the sinuses.

As time went on, other children with indefinite fever, or with gastro-intestinal symptoms, many with chronic bronchitis, many with nephritis, proved to have sinusitis. The children are X-rayed, the nose and throat carefully examined, especial stress being made on enlargement of the turbinates, lymphoid hyperplasia of the posterior pharyngeal wall and, of course ,a purulent discharge in the nasopharynx. Transillumination of the sinuses is of less value than the X-ray until the children are older. We are now studying the etiology, especially facial measurements for physical type, diet, for vitamin content and so on, and the relation to previous disease and to disease of the adenoids and tonsils. It has interested me very much because I have been interested in pulmonary fibrosis for a long time. How chronic sinusitis can cause chronic pulmonary fibrosis is a subject of speculation. There are three theories: first, it has been suggested that the discharge drains down into the lungs from the sinus while the child is asleep. I do not see how the discharge from the sinus can get past the larynx into the lungs without waking the child with coughing. Another theory is that these cases, coughing up a large amount of purulent secretion as they do, may infect the sinuses secondarily. The third theory is that they both come from a common cause. Perhaps this is a better explanation than either of the others.

I cannot refrain from speaking of one aspect of the paper on otitis, namely, to reinforce the recommendation made by Dr. LaFetra to use the electric otoscope. I cannot but feel that the pediatrician sees more of the drum of the small infant than does the man with head mirror and speculum. I used a head mirror and speculum for years before the otoscope was on the market, but I am sure that we can see more with the magnified, well lighted view given by the electric otoscope.

I should also like to take up the question of reincision of the drum in otitis media. The otologist says that one incision is enough. I do not doubt that this is true in adults. But it must be remembered that the incision in an infant is only a small fraction of an inch long, and taking into consideration the attempt of nature to heal wounds, in spite of the presence of a purulent discharge, it is quite certain that ears do sometimes heal too soon. I am sure that reincision

of the drum will sometimes prevent infection of the mastoid because I have done it many times, with a happy result.

I also wish to bring up the question of mastoid operation in small babies. We have in Bellevue many infants with acute otitis media, who have gastro-intestinal conditions, who are undernourished, or have other conditions, such as pneumonia, etc. The ear department formerly would not operate on these cases until we could get the infant into proper physical condition to withstand the shock of operation. But we could not get them into condition for the ear operator, because the systemic condition itself is due to the poor resistance resulting from the infection present, and will persist until that infection is cleared up. As a result the child often died between the two professions. Since the recommendation by Marriott of simple antrotomy with a local anesthetic, we have had a large number of these infants operated on with recovery. They would never have been able to stand a radical operation.

We have heard this evening a good deal about the importance of the treatment of the nose, but not what it is. At Bellevue, we use instillation of adrenalin and ephedrin, with the head in the vertical inverted position. In the horizontal position, the fluid goes into the floor of the nose, and not where we want it. The child should be placed with the head over the end of the table and the vertex directed towards the floor, so that the fluid goes up into the superior meatus, then to the upper part of the nasopharynx. We treat the sinuses and otitis with great success with this method.

Another question not brought up tonight, I should like to say a few words about: namely, regrowth of the tonsil. It is a common saying that if the tonsil is entirely removed, it will never grow back again. If the tonsil is taken out in small children under two, I believe it can regrow. The tonsil is merely a lymphoid infiltration of the submucosa. It is quite possible for a new infiltration of lymphoid tissue to take place, and new tonsil to grow. I have in my office several tonsils removed from small children, who now have quite good new ones. The new growth is usually flat and not as easily infected as the irregular tonsil. I beg the nose and throat men and the pediatricians not to say the tonsil was badly taken out when they see lymphoid tissue in the fossa, if the tonsil was taken out when the child was very young.

SECTION OF OTOLOGY.

Meeting of March 14, 1930.

A New Operation for Chronic Purulent Mastoiditis. Dr. J. Morrisset Smith.

Published in full in THE LARYNGOSCOPE, *August, 1930.*

DISCUSSION.

DR. JOHN R. PAGE: The convincing part of Dr. Smith's discourse is the cases he presents. He has certainly gotten excellent results. As he was particular to state, this operation is not applicable in all cases. The same is true of all the other operations, from modified radicals to ossiculectomies. To be successful, they have to be performed on properly selected cases. No one operation is suitable for all types of O.M.P.C. I was interested to hear about this operation from Dr. Smith some time ago and had him send me a description of it. Two days after I read the description, I had occasion to do a radical mastoid operation. In every case, I start with a simple mastoid operation and do only what I am led to do by the conditions found. In the case referred to, a radical mastoid was indicated, and I attempted to do Dr. Smith's operation but had to write him next day that I was unable to clean out the tympanum to my satisfaction without lowering the posterior wall and widening the canal. I mentioned that in my experience an important factor in securing a dry and thinly lined cavity was an enlarged canal and flat cavity with

removal of any projection on the anterior wall so that the tubal region could be brought to view during the after-treatment. This I could not do to my satisfaction using Dr. Smith's method. He doubtless could have on this case, but unaccustomed to his procedure, it seemed to me that the boy would have a better result if I did the radical operation. This proved to be true. He is here tonight, and he has a cavity which is a "self-cleaner", and for this reason is free from one of the objections to the radical mastoid operation that Dr. Smith makes in advocating his operation in place of it. More radical operations can be made "self-cleaners" if that idea is kept in mind at the time of operation. If the posterior canal wall or facial ridge is lowered to the full extent, so that nothing can be held back posterior to it, the epithelial debris will fall out through the meatus. Of course, where a cup-shaped tip of the mastoid is left, debris in this region will accumulate. This can be avoided, however, by the complete removal of the tip and the ledge of the diagastric fossa. The neck muscles will then fill up this region, and while there is more of a depression it is not noticeable because it is not a sharp depression, but slopes in gradually from the occiput and neck.

I said in my letter that I could understand how good results would be obtained in many cases by not such careful attention to the tympanic region as was usual in the old radical operation, just as many ossiculectomies got well without further attention to the tympanum, and had I attempted his operation in this case it would have amounted to little more than an ossiculectomy with exenteration of the mastoid. I closed by saying that I had done my usual radical operation and would pray for a "self-cleaning" cavity, and this I was fortunate in getting.

I am sorry not to have discussed Dr. Smith's operation in detail, but it boils down to this: that judgment is required in selecting cases for all these operations, from ossiculectomies to this operation. George Tobey, of Boston, gets excellent results in many cases by doing an ossiculectomy where others would do a more extensive mastoid operation. I do not think an ossiculectomy has been done at the New York Eye and Ear Infirmary or at the Manhattan Eye, Ear and Throat Hospital in the past 15 or 20 years. I never did one in my life, though I have seen several good results from them. When I was at the New York Eye and Ear Infirmary the radical mastoid was the thing everyone was doing, and we learned then to do a good operation, for we saw many done. We have heard the radical mastoid operation damned by many people who lacked interest in dressing their own cases and the vigilance necessary to keep a radical cavity clean and free from granulations. Those who appreciate the importance of these details get good results; the others do not, and they still don't know why. In selected cases, I like the modified radical, but I realize its limitations, and now this operation of Dr. Smith's, which I have tried only once and failed on, I will try again on a case that I think is suitable for it. I do not entirely agree with Dr. Smith on the question of hearing obtained in radicals. The question of hearing depends on the care of the internal wall of the tympanum and a thin lining over it. One has better access in the old radical operation to enable him to accomplish this result than in Dr. Smith's. I know of many radical mastoid cases, the hearing in which is excellent. Just as good hearing can be obtained in some radicals as in some modified radicals. It depends on the selection of cases and the careful after-treatment.

I congratulate Dr. Smith and hope to have an opportunity to try again his operation on a suitable case.

DR. GOTTLIEB: Dr. Smith did not say anything about the indications for the operation. It is generally accepted by otologists that the indications for the radical mastoid operation are danger to intracranial contents, such as labyrinthine disturbances, facial paralysis, headache—and not the presence of aural discharge which has continued for a long period. As I understand it, the operation in which this new procedure was used was performed because of the presence of a running ear alone, and not because the intracranial contents were in danger. It is quite possible that Dr. Smith implies that the indication for this new procedure is the presence of an annoying discharge from the ear of long standing, and for the purpose of obviating the more formidable radical mastoid operation.

DR. J. MORRISSET SMITH (closing): I do not think there is any comparison between an ossiculectomy and this operation; ossiculectomy simply means the removal of the malleus and the incus through the external auditory canal, while this operation not only removes the diseased tissues from the middle ear and attic but from the antrum and mastoid as well. A long-continued, foul-smelling discharge from the middle ear, persisting in spite of local treatment and medications, means necrosis of the bone within the mastoid. An operation should be done to check the progress of the infection. Operation in any case is not indicated to check the discharge but to remove the danger. Waiting for severe headaches or other symptoms of an intracranial extension of the infection before operating is certainly not advisable. Operation should be performed before these symptoms occur, if possible.

As stated in the article, this operation should not be attempted where there is a small, deep external auditory canal as there is not sufficient space to properly perform the different steps or to carry out the necessary after-treatment.

In regard to hearing following the radical operation: Having performed a large number of the radical operations during the last 15 years and having followed some of the cases over that period of time, I must confess my disappointment at the practical hearing results obtained. By this I mean that I have been unable to obtain enough hearing to be of practical value to the patient in the ordinary routine of life. Two of the patients presented tonight had double mastoid operations. The first, a radical on one side, and a new radical operation on the other.

The second, a radical operation on one side and a complete simple on the other. In both instances the hearing averaged 16 per cent better by the audiomter in the ears upon which the radical operation had not been performed. I am convinced better hearing results will be obtained with the new radical operation. As I pointed out in my article, the postoperative hearing depends largely upon the epidermatization of the internal wall of the middle ear without granulation tissue. This new radical operation has no postoperative radical cavity or flap, simply a middle ear and normal external auditory canal, making it easier, I believe, to keep the internal wall free from granulations.

The operation is not as easy to perform as the radical, due to the fact that the posterior canal wall is allowed to remain in position, making it more difficult to remove the diseased structures from the middle ear. I wish to again repeat that the operation is presented as an effort to improve the results obtained in operating upon a certain number of the chronic cases.

Masking Effect of an Interfering Tone on a Deafened Ear. Dr. John Guttman and Dr. L. B. Ham.

Published in full in THE LARYNGOSCOPE, *September, 1930.*

DISCUSSION.

DR. E. M. JOSEPHSON: I was very much interested in Dr. Guttman's paper, especailly in his hypothesis as to the innervation of the accommodative middle ear mechanism. It was suprprising to hear him say that he thought that the afferent tract for the accommodative reaction was the cochlear nerve. If that were true, it is obvious that the mechanism would be futile as a protection to the inner ear mechanism. The overloud sound would have to reach and damage the inner ear mechanism and the cochlear nerve before the protective reaction would be under way. It appears far more probable, as well as reasonable, to assume that the afferent tract of the accommodative reaction of the middle ear mechanism lies along the nerves which innervate the tympanum, the ossicles and other structures of the middle ear, and the efferent or motor tract along the nerves which supply the tensor tympani and stapedius muscles.

In this connection it would be interesting to mention a case which I have seen this very afternoon, who has been suffering for the past two months with a lower-half headache and neuritis involving the left supra- and infraorbital nerves. She came to see me because in the 24 hours immediately preceding she had suddenly become deafened, especially in the left ear. She had also had echo-hearing—diplacusis. Fortunately, I had had the opportunity to examine her ears several months prior and had found them normal. On examining them

today I found retraction and immobility of the left drum, similar to that which we so frequently find in chronic deafness. Catheterization and politzerization of the ear failed to relieve the retraction or the deafness. After application of the negative pole of the galvanic current to the ear, the retraction was promptly relieved, the drum became freely movable upon Valsalva inflation, and the hearing improved considerably. I have frequently found it possible, almost regularly, to relieve the chronic retraction of the drum in cases of C.P.D. by galvanization. This makes it appear to be probable, if not certain, that these retractions are often the result of spasm of the tensor tympani and stapedius muscles, rather than due to obstruction of the Eustachian tube; and this view is confirmed by the repeated finding of atrophic, widely patent tubes in these cases, instead of the generally hypothesized Eustachian obstruction. This case also indicates the possibility and probability that the afferent tract of the accommodative reflex of middle ear being bound up with the fifth and other sensory nerves of the head.

Speaking of the relief of retraction of the drum membrane by galvanism brings to mind another matter—the serious damage wrought by persistent inflation of the middle ear in these cases of chronic deafness with drum retraction. Though the retraction can be frequently relieved by galvanization, inflation is usually futile in these cases and merely serves to stretch and damage the structure of the tympanic membrane. This results in a marked loss of the lower tones, which contrasts markedly with the recovery of the lower tones, which often follows upon relief of the spastic retraction of the eardrum by galvanization without prior inflation.

It scarcely seems possible that Dr. Guttman would convey the impression that he feels that the objective of the middle ear accommodative mechanism is to transmit the full volume of the intense sounds to the inner ear. Such a state of affairs would be disastrous to the delicate inner ear mechanism. It is far more probable that it serves in much the same role as does the sound control mechanism of phonographic and other sound-recording devices—to prevent the amplitude of vibration rising above a certain fixed maximum. It appears probable that our perception of sound intensity is a composite sensation of cochlear sound plus the deep sensation of adjustment of the accommodative mechanism of the middle ear.

At this point, I wish to raise objection to our classification of the deafnesses. We speak of conductive and perception deafnesses, applying perceptive to all other than the conductive type of cases. Such terminology is erroneous. Psychology applies the term deafness to mental interpretation of sensation. Therefore, we must divide off our sensation deafnesses, due to injury of the sensory mechanism, from perceptive and apperceptive (tone and word) deafnesses. Our classification should be as follows: Conductive, sensory, perceptive and apperceptive deafness. Unfortunately, we know comparatively little about some of these types, for hitherto attention has not been definitely directed to them.

As regards to the masking effects of one tone upon the other, Dr. Guttman's very scholarly paper leaves one in some doubt. For normal individuals, a decibel does not at all ranges constitute a unit of actual sensation. The decibel is more truly a power unit, which only approximately and as an occasional average corresponds to a unit of sensation. In other words, not many normal ears can differentiate a sense of variation of loudness with a change in power of one decibel. Nor is the ratio of decibels to sensation constant for the different frequencies, even in the same individual, nor is it constant from day to day. Concerning the ratio of decibels to sensation units in the deafened individuals, extensive data is lacking. Consequently when Dr. Guttman and his collaborator raised the power of their two tones by 20 or 50 decibels from the threshold figures, we are at loss to know if the loudness of the tow tones was increased by equal amounts in the normal and in the various types of deafness. There are two possibilities underlying the masking effects reported by Dr. Guttman. One is that the sensation-decibel ratios in the deafness cases are such for the lower tones that an increase of a definite number of decibels does not result in as great an increase in the intensity of those tones as in the intensity of the higher tones. The other possibility is that which has been

offered in the past, i. e., that the lower wave length may set the auditory mechanism in vibration, overcoming the resistance which it offers to the higher tone waves—the lower frequency acting to all effects and purposes as a carrier wave for the higher frequencies.

R. L. WEGEL (Bell Telephone Laboratories): This paper of Dr. Guttman and Dr. Ham is rather interesting, in that it appears to be the start of a new type of investigation in the pathology of hearing.

About 10 years ago Dr. Guttman was one of the first to apply the vaccum tube in making an audiometer, but apparently he did not follow his work far enough to make us acquainted with the type of apparatus in order to give data on hearing by means of its use. The vacuum tube audiometer has since been developed into a satisfactory piece of apparatus to be used in diagnosing, and it appears to be gradually replacing the tuning fork. It is rather interesting in this connection that although the audiometer and the audiogram have a number of other uses or advantages, from the standpoint of diagnosis it is now used only to give the same information as was formerly obtained from the tuning fork. The old test by means of the tuning fork for air conduction of the lower tone limit corresponds in the audiogram to an indication of lowered acuity at low frequencies and is taken to be indicative of the conductive deafness. Tests with the Galton whistle or monochord for air conduction were taken as indicative, when the ear was insensitive to these instruments, of nerve deafness. This same information is obtained from the audiogram. I understand that the correlation of audiograms with histological investigation at Johns Hopkins is beginning to demonstrate that there is, at least in cases of mild deafness, a correlation between the depressions in the audiogram curve and the position of a lesion in the organ of Corti and that the usual depression which occurs in the neighborhood of 4,000 to 8,000 double vibrations corresponds to a lesion in the organ of Corti at a distance of 4 to 8 m.m. from the round window. It is remarkable, if true, that nerve lesions always begin at these places.

Dr. Guttman proposes the introduction in the research on the subject of a type of measurement. While it is not strictly correct to call this a measurement of paracusis, this term comes nearer to describing the effect than any other single term that I can think of. This is of course only one more of a large number of tests which might be made. We have in the audiometer a test of acuity. Dr. Guttman proposes a measurement of paracusis. We could also investigate pitch sensibility, intensity sensibility, distortion in the ear which is exaggerated in abnormal cases and recognized by a patient when he says that he hears echoes and sounds which he knows do not exist, tests and measurements of tinnitus, and so on.

Taking Dr. Guttman's measurements in particular, the question immediately arises: What use can be made of them? Two possibilities are immediately suggested: the first is to use it as a test in diagnosis, and the second is to apply it in a more fundamental program of the study of the mechanism of hearing. As a routine diagnostic test it seems to have comparatively small possibilities on account of the elaborate nature of the apparatus and the time which would be consumed ordinarily in making it. It seems, therefore, that the principal value of such a test is in its bearing on the more general study of hearing, leading us to a more comprehensive understanding of the subject and so, indirectly, to better diagnostic tests.

I gather from hearing this talk that the authors of the paper have definitely measured a "paracusis" in many abnormal ears. With the assumption that this is a true finding, Dr. Guttman proceeds to justify it on the assumption that it depends on the reduction in the ability of the apparatus of accommodation to function; that is, due to a pathological condition of the tensor tympani and the stapedius muscle, or possibly to an ankylosis of some kind. This appears quite reasonable in view of the fact that the magnitude of the effect which he observes seems never to be greater than about 10 sensation units, which corresponds to stiffness changes of the apparatus in the ratio of about one to three. If this explanation is correct, the effect should be most noticeable at very low frequencies, say below 1,000 decibels, and very little effect should be observed at the higher frequencies. The measurements seem to indicate the effects at all

frequencies but data is not yet sufficiently well worked out to make it safe to draw any specific conclusions.

The authors are to be congratulated on their work so far and it is to be hoped that they will improve their apparatus and obtain sufficient data along this line to permit of the drawing of more specific conclusions.

DR. GUTTMANN: I wish to thank Mr. Wegel for coming here and discussing our paper, and especially for the statement that I was the first to devise and describe the audiometer based on the vacuum tube principle, and that its practical usefulness is greatly exaggerated. The judgment of a man of Mr. Wegel's experience is of great importance.

We have never considered our work as an additional practical functional hearing test; our endeavor was to find out and to explain something which was probably not so well known before. Our conclusions were found to be correct not only concerning the low, but also the high tones. Regarding paracusis Willisii, we found it to be present in perceptive deafness as well as in conduction deafness. Formerly it was thought to be present only in the latter.

Our explanation of paracusis is found in the lessened differential threshold due to increasing deafness, in addition to the fact, as assumed by previous authors, that the low interfering voices are not heard by an ear suffering from conduction deafness.

In reply to the last speaker, I wish to emphasize the fact that more energy is needed for the reflex action, suppressing the perception of the interfering tone than for the perception of the primary tone.

BOOK REVIEW.

Modern Otology. By Joseph Clarence Keeler, M.D., F.A.C.S., Professor of Otology, Jefferson Medical College; Otolaryngologist, Germantown Hospital; Consulting Otolaryngologist, Pottstown Hospital, Pottstown, Pa., and Newcomb Hospital, Vineland, N. J., etc. With 858 pages, 90 original illustrations and 15 colored plates. Philadelphia: F. A. Davis Company, 1930. Price $10.00 net.

This textbook was inspired, according to the author's preface, by the occasional dissatisfaction of the existing textbooks. The author has attempted to correct and bring up-to-date the teachings of otology which he feels should be available to the medical profession.

This volume is dedicated to the late Dr. S. MacCuen Smith, with whom the author was closely associated for a period of more than 25 years. The work itself is very nicely gotten together, being divided into anatomic, physiologic and clinical sections; each section is then subdivided into chapters pertaining to some particular subject. In this way reference can be quickly made without the usual delay of having to hunt through the book to find the point in question.

Intracranial complications are dealt with very minutely, as is the labyrinth. Near the end of the book there is a special section devoted to Otology in Children; there is a chapter on Deaf Mutism, and the final chapter is one on the Medico-Legal Aspects of Otology.

The book is printed in pleasant, large type and is very generously supplied with clear and explicit diagrams and photographs.

In the reviewer's opinion, Keeler's Modern Otology is bound to take its place with the recognized standard textbooks in every day use by both specialist and student.

M. F.